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SENSORY CHANGES IN THE SUBACUTE COMBINED DEGENERATION OF PERNICIOUS ANEMIA *

ARTHUR S. HAMILTON, M.D. AND CHARLES E. NIXON, M.D.
MINNEAPOLIS

In the following article we desire to summarize the development of our knowledge of sensory phenomena associated with pernicious anemia and to record the sensory changes encountered in forty-one cases of pernicious anemia studied in the Elliott Memorial Hospital of the University of Minnesota and in the university service of the Minneapolis General Hospital. Nearly all these cases were also studied by the members of Dr. Rowntree's staff in internal medicine and no case appears in our list in which the diagnosis of pernicious anemia was disputed. Most of our cases belong to the Addison-Biermer type of pernicious anemia.

HISTORICAL SURVEY

Addison [1] (1855), in his original description of pernicious anemia, said nothing of involvement of the nervous system except that he found some fatty degeneration of a portion of the semilunar ganglion of the solar plexus.

Biermer [2] (1872) referred to: weakness, giddiness and palpitation and also reported capillary hemorrhages in the brain and the retina.

In 1880, Herbert W. Little [3] stated: "The indications of the nervous affection in this disease are: frequent cephalalgia, vertigo, syncope, neuralgic pains, dyspepsia, vomiting, diarrhea, mental depression, muscular weakness, pallor of the skin, reduction or rise of temperature." The author thought the nervous system the primary seat of trouble and involvement of the vasomotor system important.

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* From the Division of Nervous and Mental Diseases and the Neuropathological Laboratory, University of Minnesota Medical School.

[1] Addison, T.: On the Constitutional and Local Effects of Disease of the Suprarenal Capsule, 1855.

[2] Biermer: Progressive perniciöse Anaemie, Cor.-Bl. f. schweiz. Aerzte **2:** No. 1, 1872.

[3] Little, H. W.: Progressive Pernicious Anemia, a Disease of the Vasomotor System, Med. Rec. **17:**313, 1880.

In 1884, Lichtenstern[4] reported two cases of pernicious anemia in tabetics. It is probable that these were not cases of tabes but of pernicious anemia. Lichtenstern referred to posterior column degeneration, but said nothing of sensory changes.

In 1887, Lichtheim[5] reported three cases of anemia; one patient had stiffness of the legs, a feeling of weakness, paresthesia, ataxia and the Romberg sign. There was widespread degeneration of the columns of Goll and Burdach, but of the latter a small rim remained intact. Foci of degeneration were found in the lateral and anterior columns. The peripheral nerves were not affected.

In Putnam's[6] (1891) eight cases of system sclerosis, the nerve roots were more or less altered in the four in which pathologic studies were made, the anterior somewhat more than the posterior. In all of his cases, numbness was a pronounced feature, lancinating pains in only one. One patient complained of a sense of constriction around the chest and abdomen and that at times she felt "as if her stockings were filled with pebbles." Objective impairment of muscle sense and diminished cutaneous sensibility were observed in all but one case.

Dana[7] (1891) reported a patient with a sensation of pins and needles in her heels and queer, indescribable, somewhat painful feelings in both lower extremities with every change of weather. There was no cutaneous anesthesia. The posterior roots in the lumbar region showed decided degenerative changes.

Grainger Stewart[8] (1891) reported a case which he believed conformed to the Putnam-Dana type of combined sclerosis. There was painful tingling in the upper and lower extremities. A year later the paresthesias were more marked and sensibility was diminished.

Von Noorden[9] (1891-1892) found paresthesias in the extremities and impaired sensibility in the feet, and noted an intact area next the posterior roots though the posterior columns were greatly degenerated.

---

[4] Lichtenstern, O.: Progressive perniciöse Anämie bei Tabeskranken, Deutsch. med. Wchnschr. **10**:849, 1884.

[5] Lichtheim: Zur Kenntniss der perniciösen Anämie, Verhandl. d. Cong. f. inn. Med. **6**:84-99, 1887.

[6] Putnam: A Group of Cases of System Sclerosis of the Spinal Cord, Associated with Diffuse Collateral Degeneration Occurring in Enfeebled Persons Past Middle Life, and Especially in Women; Studied with Particular Reference to Etiology, J. Nerv. & Ment. Dis. **16**: (Feb.) 1891.

[7] Dana, C. L.: Subacute Combined Sclerosis of the Spinal Cord and Its Relation to Anemia and Toxemia, J. Nerv. Ment. Dis. **26**:1, 1889; 1891, February and April.

[8] Stewart, Grainger: Notes on a Case Probably Referable to a Recently Described Form of Spinal Disease, Brit. M. J. **1**:1219, 1891.

[9] Von Noorden: Untersuchung über schwere anämie, Charité Ann., 1891, p. 217.

Eisenlohr[10] (1892) described a case with itching; there was degeneration of the right saphenous nerve and subacute combined degeneration of the cord.

Minnich[11] (1892) reported six cases of pernicious anemia, in which all the patients had objective sensory disturbances, increasing toward the periphery, such as loss of position sense in toes, lowered pain sensibility, especially in the peripheral parts, but not so involved as tactile sense, together with impaired position sense and some impairment of the sense of pressure over the tibia. The cords in these cases showed the usual patchy degeneration in Burdach's column and sharply defined areas of degeneration in Goll's column. The posterior roots and the peripheral nerves, where examined, were normal.

Nonne[12] (1893) reported two cases of pernicious anemia with numbness of the hands and feet and girdle sensation. In one case the numbness was present long before the anemia was obvious. There were some hypalgesia and hypesthesia. In a report[13] (1895) of seventeen cases of pernicious anemia with cord changes he noted sensory disturbances in four. The peripheral nerves were examined in three and no noteworthy degeneration found. With the Weigert method, the posterior roots and root entrance zones appeared practically intact, but with the Marchi method there was some degeneration of the posterior roots.

Burr[14] (1894) stated that though paresthesias are frequent and delayed or diminished sensation not infrequent, absolute anesthesia almost never occurs. He comments on the marked discrepancy between the severe anemia and the normal cord in one case. The peripheral nerves in one case were normal.

Boedeker and Juliusberger[15] (1896) reported the case of a patient with pain in the entire body, especially the bones, and a sense of deadness in the feet. In a second patient there was a feeling of deadness in the legs, coldness and numbness in the fingers and later a hyperesthesia of the entire body and tearing pains in the legs. Three months later there was disturbance of the muscle sense with practically intact skin sensibility and two months after that, a feeling of deadness in the legs with essentially still intact skin sensibility, including temperature sense. One month later the muscle sense was practically gone. Skin sensibility was lost up to the neck. There was sclerosis of the middle root zone but especially of Goll's column and also of a part of the root entrance zone. The extramedullary roots and peripheral nerves were intact.

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[10] Eisenlohr: Ueber primäre Atrophie der Magen- und Darmschleimhaut und deren Beziehung zu schwerer Anämie und Rückenmarkserkrankung, Deutsch. med. Wchnschr. **49**:1105, 1892.

[11] Minnich, W.: Zur Kenntniss der im Verlauf der perniciösen Anämie beobachteten spinal Erkrankungen, Ztschr. f. klin. Med. **21**:25-60, 264-314, 1893; **22**:60-86, 1893.

[12] Nonne, M.: Beiträge zur Kenntniss der im Verlaufe der perniciöser Anämie beobachteten Spinalerkrankungen, Arch. f. Psychiat. **25**:421-449, 1893.

[13] Nonne, M.: Weitere Beiträge zur Kenntniss der im Verlaufe letaler Anämien beobachteten Spinalerkrankungen, Deutsch. Ztschr. f. Nervenheilk. **6**:313-368, 1894-1895.

[14] Burr, C. W.: The Spinal Cord Lesion and Symptoms of Pernicious Anemia, Univ. Med. Mag. **7**:472-491, 1894.

[15] Boedeker and Juliusberger: Demonstration von Rückensmarksveränderungen bei tötlicher Anämie, Centralbl. f. Nervenheilk u. Psychiat. **7**:315-318, 1896.

Lloyd's[16] (1896) patient complained of cold sensations running up and down his legs, of pricklings in the same regions and of a feeling of walking on cotton; also girdle sensations about the abdomen. The lesion in the posterior columns was about midway between the columns of Goll and Burdach, extending not quite to the posterior commissure in front, nor quite to the periphery of the cord behind. The posterior root zone was not seriously involved. The nerve trunks showed some change with the Weigert stain.

Bastianelli[17] (1896) reported a group of patients with subacute combined degeneration symptoms developing first or coincidental with the anemia, which is of secondary importance. These cases he seems to set aside from true subacute combined degeneration of pernicious anemia, but it is probable that in at least some the symptoms of pernicious anemia would appear later.

Petren[18] (1896) found loss of touch, temperature and muscle sense in all the extremities, especially the legs, in one of his patients.

Dana[19] (1899) referred to the prominent and persistent subjective sensory phenomena and found diminution of touch, pain and temperature sense.

Russell, Batten and Collier[20] (1900) found girdle sensation and severe lightning pains to be frequent. Anesthesia of the lower extremi-

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[16] Lloyd, J. H.: The Spinal Cord in Pernicious Anemia, J. Nerv. & Ment. Dis. **21**:225-235, 1896.

[17] Bastianelli, G.: Le sclerosi combinati del midollo spinale nelle anemie perniciosi, Bull. de roy. Acad. de med. di Roma **22**:197-244, 1896-1897; Ref. in Neurol. Centralbl. **16**:78, 1897.

[18] Petren, K.: Bidrag till Kännedomen om Ryggmärgsförändringar vid Perniciös Anämie, Nord. Med. Arkiv. **6**:71, 1896; Ref. Neurol. Centralbl. **15**:747, 1896.

[19] Dana, C. L.: Subacute Ataxic Paraplegia and Combined Sclerosis—a Form of Spinal Disease Associated with Lethal Anemia and Toxemia, Med. Rec. **55**:897-902, 1899.

[20] Russell, J. S. R.; Batten, F. E., and Collier, J.. Subacute Combined Degeneration of the Spinal Cord, Brain **23**:39-110, 1900.

ties and lower trunk rapidly becomes absolute and rises segment after segment.

Billings[21] (1902) reported thirty-six cases of subacute combined degeneration with pernicious anemia, in all of which paresthesia occurred early. In twenty-four the sensory changes were subjective only: tingling, numbness, coldness, formication, heat, etc. Hyperesthesia occurred in one case. Girdle sensation was present in all developing spasticity. The subjective symptoms were usually stationary, but disappeared in one case. In some there was complete loss of sense of position even though touch and temperature were only slightly involved. The column of Lissauer and the posterior roots were intact.

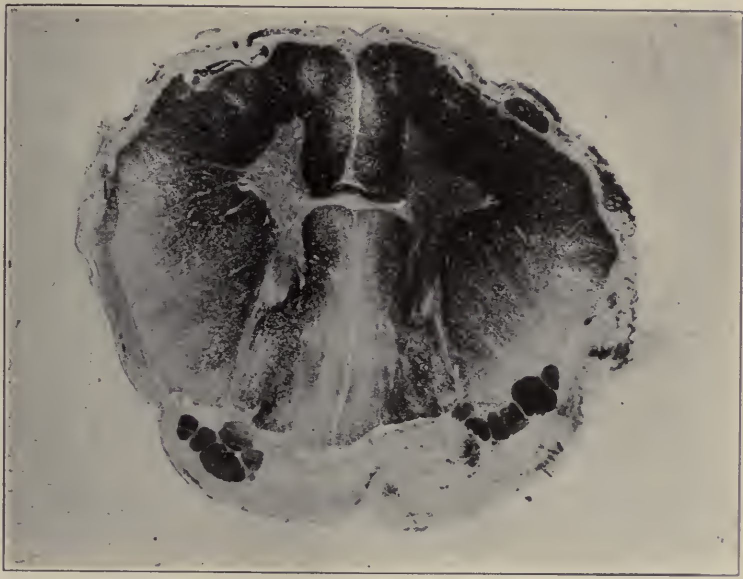

Fig. 1 (Case 2).—Third cervical segment cord; Weigert stain.

J. M. Clark[22] (1904) stated that the most prominent clinical symptoms are paresthesias of various kinds. A common complaint was of numbness or deadness of the fingers which rendered fine movements difficult. These paresthesias vary much in intensity during the course of the illness. Objective disturbance of sensibility is not marked, but in the later stages may consist of some deficient appreciation for all forms of sensation over the lower extremities. In one case examined the peripheral nerves were healthy.

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[21] Billings, F.: The Changes in the Spinal Cord and Medulla in Pernicious Anemia, Boston M. & S. J. **147**:225-233, 257-263, 1902.

[22] Clark, J. M.: On the Spinal Cord Degenerations in Anemia, Brain **28**:441-459, 1904.

Putnam and Taylor [23] (1901) reported some form of paresthesia as strikingly prominent and usually felt first in the feet and then in the hands, either in all the fingers alike or in the median or ulnar areas by preference. The proximal segments of the shoulder or thigh were at times attacked early, if not first, as occasionally in tabes.

Bramwell [24] (1915) insisted particularly on the occurrence of cord symptoms which may long antedate positive signs of pernicious anemia—in one about three years. Then rapid destruction of blood takes place. In one patient there were shooting pains in the legs, numbness and tingling in the feet and a cold feeling in the knees and elbows. Bramwell also reemphasized the stocking and glove distribution of the sensory loss.

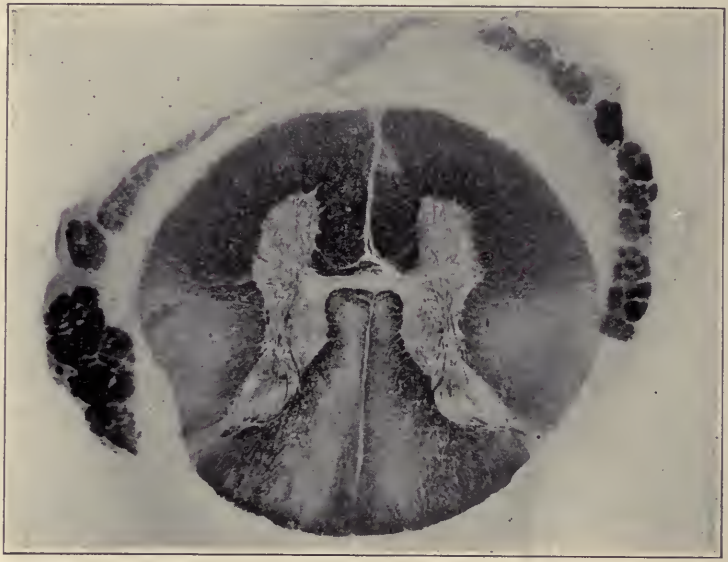

Fig. 2 (Case 2).—Twelfth thoracic segment cord; Weigert stain.

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[23] Putnam, J. J., and Taylor, E. W.: Diffused Degeneration of the Spinal Cord, J. Nerv. & Ment. Dis. **28**:1-21, 1901.

[24] Bramwell, B.: On the Association of Pernicious Anemia with Subacute Combined Degeneration of the Spinal Cord, Edinburgh M. J. **14**:260-276, 1915.

In 1913 and 1914, J. Déjerine, A. Déjerine, Crouzon and Jumentie,[25] were the first clearly to point out the early and marked development of loss of deep sensibility in subacute combined degeneration. In the first publication, Déjerine divided sclerosis of the posterior and lateral columns into two groups: (1) classic tabes complicated with involvement of the lateral columns. In this type the sensory changes are a marked alteration of the superficial senses. There is a slowing rather than a diminution in the intensity of the perception of the latter. There exists also extensive alteration of deep sensibility. (2) A second variety of combined sclerosis of subacute course shows no signs of syphilis, and the condition is rather associated with pernicious anemia. There is integrity of all kinds of superficial sensibility, tactile in particular, while deep sensibility as shown in sense of position, bone, deep pressure and stereognosis, is abolished.

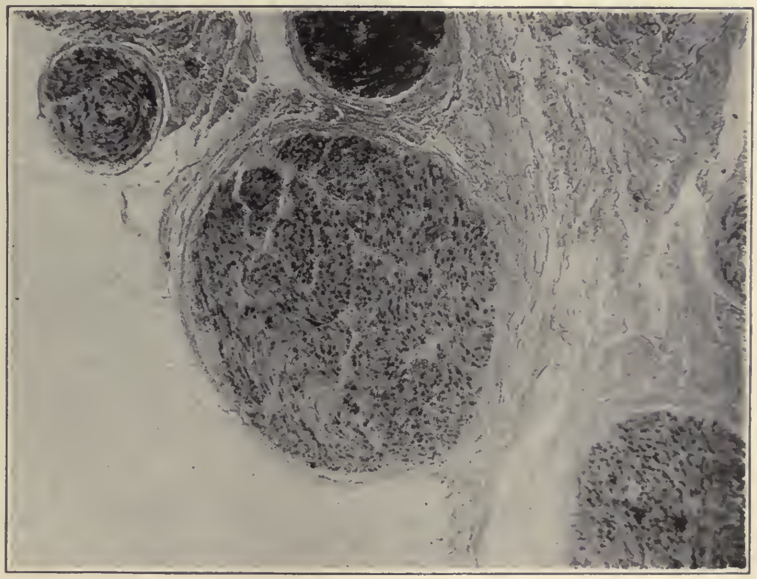

Fig. 3 (Case 2).—Peripheral nerve; Weigert stain.

The explanation of this variation lies in the difference in the topography. In the tabetic process, the sclerosis is of radicular origin. The external part of the column of Burdach, the posterior horn and the posterior roots are always involved, and since the external part of Burdach is the point of passage through the cord of the short fibers of the posterior roots, conductors of tactile, pain and temperature sense, these are naturally involved. In subacute combined sclerosis, the posterior roots and the external part of the column of Burdach are always intact, and only the long fibers of the columns of Goll and the adjacent part of the column of Burdach are involved. Since the different kinds of deep sensibility pass only by the long fibers of the posterior columns, these sensibilities are earliest and most involved in subacute combined degeneration of the cord. Déjerine has called integrity of superficial sensibility with marked disturbance of deep sensibility the syndrome of the long radicular fibers of the posterior columns.

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[25] Déjerine, J.: Le syndrome des fibres radiculaires longues des cordons postérieurs, Compt, rend. Soc. de biol. **75**:554-556, 1913. Déjerine, A. and Mouzon, J.: Contribution a l'etude du syndrome des fibres radiculaires longues des cordons postérieurs dans l'anémies pernicieuse, Rev. neurol. **22**:382-388, 1914. Déjerine, J. and Jumentie, J.: Un cas de syndrome des fibres radiculaires longues des cordons postérieurs, suivi d'autopsie, Rev. neurol., ibid., 271-273.

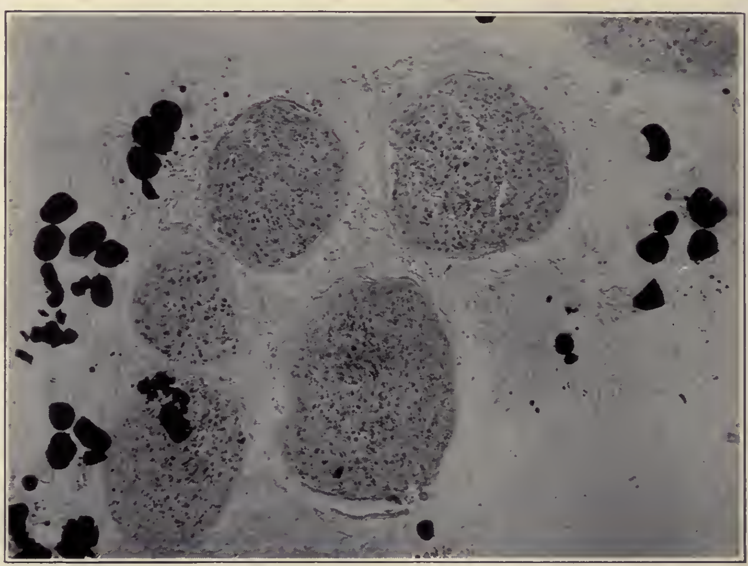

Fig. 4 (Case 2).—Peripheral nerve; Marchi stain.

Cadwalader[26] (1916) reported eight cases recently observed by himself. All complained of subjective disturbances of sensation. Tactile, pain and temperature sensibility were normal in all but one case, while vibration sensibility and sense of position were diminished or lacking in all.

Schaller[27] (1918) concluded that "the cord changes in combined sclerosis are pseudo-system degenerations of toxic type." He explains the early involvement of deep sensation and the conservation of superficial sensation by the localization of the lesions.

---

[26] Cadwalader, W. B.: Diagnosis of Subacute Combined Sclerosis of the Spinal Cord Associated with Pernicious Anemia, J. A. M. A. **66**:2035-2036, 1916.

[27] Schaller, W. F.: Spinal Cord Changes in Combined Sclerosis, California State J. M. **16**:44, 1918.

Woltmann[28] (1919) reported 150 cases of pernicious anemia from the Mayo Clinic. Vibration or joint sensibility was impaired in 92 per cent. of the patients showing involvement of the nervous system, whereas superficial sensibility was impaired in only 42.4 per cent. of their cases.

SENSORY PHENCMENA

Frequency of Sensory Phenomena in Pernicious Anemia.—Authors vary widely in their opinions. McCrae[29] reported forty cases of pernicious anemia; ten patients showed nervous manifestations by examination, but only four complained of definite nervous troubles. McPhedran[30] found symptoms of disease of the nervous system in nine out of twenty-two patients and Woltmann in 80.6 per cent. of his 150 patients.

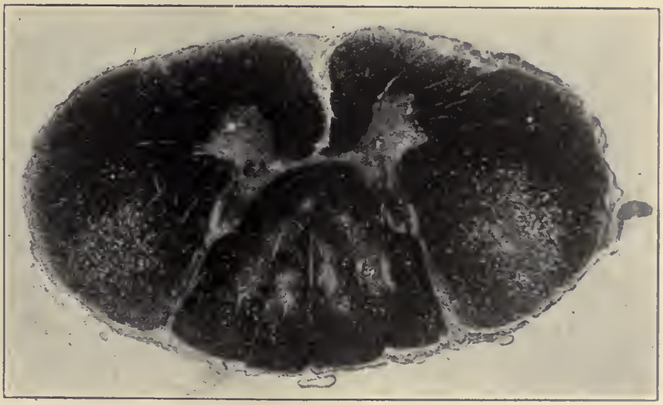

Fig. 5 (Case 3).—Seventh thoracic segment cord; Weigert stain.

Subjective Sensory Phenomena.—Subjective sensory disturbances are so common and so pronounced in subacute combined degeneration, as seen by the foregoing, that they have come to be looked on as most important diagnostic phenomena, and by some are considered so characteristic as almost in themselves to constitute a sufficient basis for diagnosis. The most frequent sensory disturbances are: tingling, numbness, sensation of deadness, stiffness, tightness, heat or cold or of something trickling or creeping and shooting pains.

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[28] Woltmann, H. W.: Nervous Symptoms in Pernicious Anemia, Am. J. M. Sc. **157**:400 (March) 1919.

[29] McCrae, T.: Pernicious Anemia. The Statistics of a Series of Forty Cases, J. A. M. A. **28**:148-150, 1902.

[30] McPhedran, A.: Observations on the Nature and Treatment of Pernicious Anemia, Lancet **1**:148-150, 1902.

These sensations are at first on the periphery of the limbs, usually in a stocking and glove fashion, more rarely in a nerve root distribution. They have been reported as appearing in the tongue. Subsequently they spread toward the trunk, and are followed at a variable interval by sensory loss.

Objective Sensory Phenomena.—From the time of Putnam[6] (1891) objective sensory phenomena have been noted, but there is little specific information as to the type of sensory loss, until the report of Minnich,[11] who states that there is a lowering of all forms of sensibility, increasing toward the periphery, with tactile sense more involved than pain and with sense of position lost in the toes. Later observers reported various degrees of anesthesia and analgesia, loss of sense of position and impaired localization.

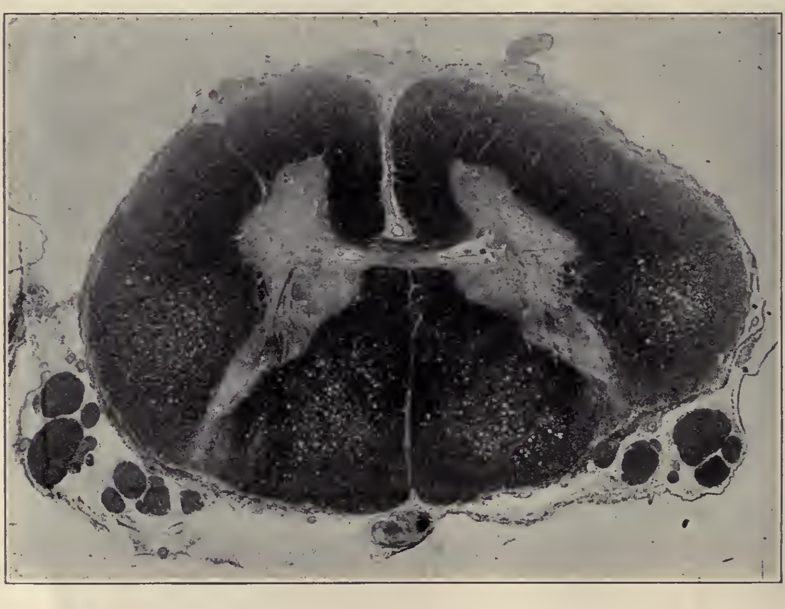

Fig. 6 (Case 3).—First lumbar segment cord; Weigert stain.

Russell, Batten and Collier[20] spoke of a considerable loss of sensibility of all forms in the legs and lower part of the trunk with sense of passive position lost in the lower extremities and much impaired in the hands. Other forms of sensibility were impaired equally and in an increasing degree. Anesthesia of the skin was always peripheral first, appearing over the feet and hands, but as it gradually extended upward it became segmental in distribution, and its upper limit on the trunk was always a segmental limit, sharply defined. The highest level they observed was the upper limit of the sixth cervical root area. In its progress upward it may be limited for a long time to a certain segmental level and then suddenly encroach for several segments higher and over the newly involved area may show marked fluctuations in its progress to complete anesthesia. Anesthesia dolorosa and delay amounting to as much as fifteen seconds have been encountered. The lower lumbar and the first sacral areas were always affected before the lower sacral and upper lumbar areas, and the lower cervical segments before the upper thoracic segments. They state that microscopically even in extreme cases there is a narrow band of normal tissue just inside the posterior horns and that the column of Goll is more affected than that of Burdach.

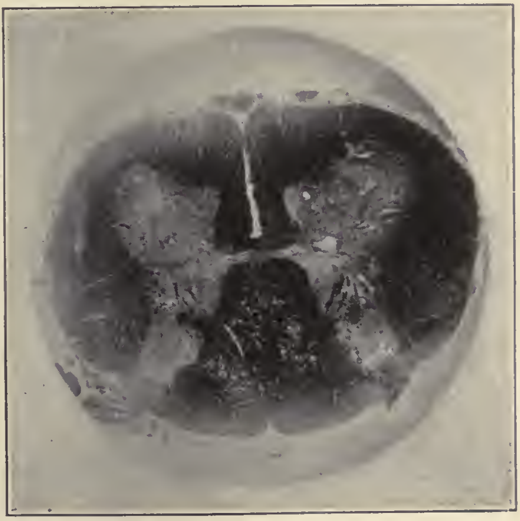

Fig. 7 (Case 3).—Second lumbar segment cord; Weigert stain.

Gordon [31] reported a case with a sensory syringomyelic dissociation in the upper extremities.

Foster Kennedy [32] reported a case with some diminution of pain and tactile sensation of irregular distribution on the legs. Temperature sense and deep muscle pain sense were normal, but there was marked loss of sense of position in feet and hands.

Hyperthesia has been noted occasionally, among others by Boedeker and Juliusberger,[15] Billings,[21] Nonne,[12] Minnich,[11] and

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[31] Gordon, A.: Histological Changes of the Spinal Cord in Pernicious Anemia Apropos of a Case with Diffused Degeneration, New York M. J. **90**:8-12, 1909.

[32] Kennedy, Foster: Subacute Combined Degeneration of the Spinal Cord Associated with Anemia, Med. Rec. **84**:84, 1913.

Thompson.[33] In one of our cases there was marked hyperesthesia throughout a period of two years; the patient objected strenuously to a blanket on her feet and legs. During an attack of anasarca, this hyperesthesia disappeared and did not return.

RELATION OF SEVERITY OF BLOOD PHENOMENA TO NERVOUS PHENOMENA

Lack of parallelism in the severity of the blood and nervous phenomena has been noted by many writers. On the basis of this lack of congruence, Bastianelli [17] divided his cases into two groups. In the first group anemia is the predominating symptom, the cord symptoms developing later and in mild degree. In the second group nervous symptoms develop first or coincidently with the anemia, and the nervous symptoms predominate throughout.

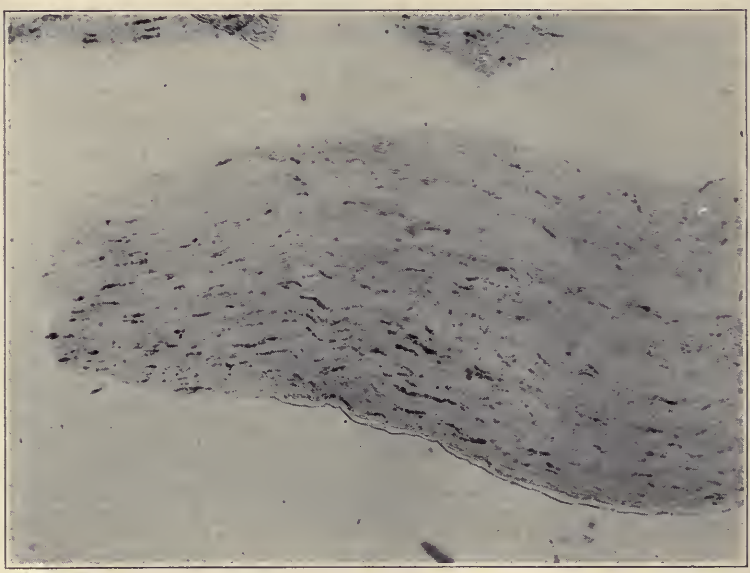

Fig. 8 (Case 3).—Posterior roots; Marchi stain.

J. M. Clark [22] said the anemia may (*a*) precede the cord changes; (*b*) it may accompany them from the first, and proceed pari passu with them; or (*c*) it may appear late in the course of the cord affection, and his statement is supported by the cases of a few other writers.

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[33] Thompson, T.: A Case of Combined Degeneration of the Spinal Cord Demonstrating the Nature of Afferent Impulses in the Posterior Columns, Brain **34**:510, 1911-12.

Siemerling[34] reported a case in which there was improvement in the blood picture with simultaneous progression of the cord involvement.

While receiving high caloric feeding the hemoglobin of one of our patients (W. G.) rose from 40 to 80 per cent. and the red cell count from 1,080,000 to over 3,000,000. This man previously had shown no impairment of vibration sense, yet during this improvement in the blood, accompanied by improvement in all his symptoms except the subjective sensory disturbances, he showed diminished appreciation of vibration for the first time.

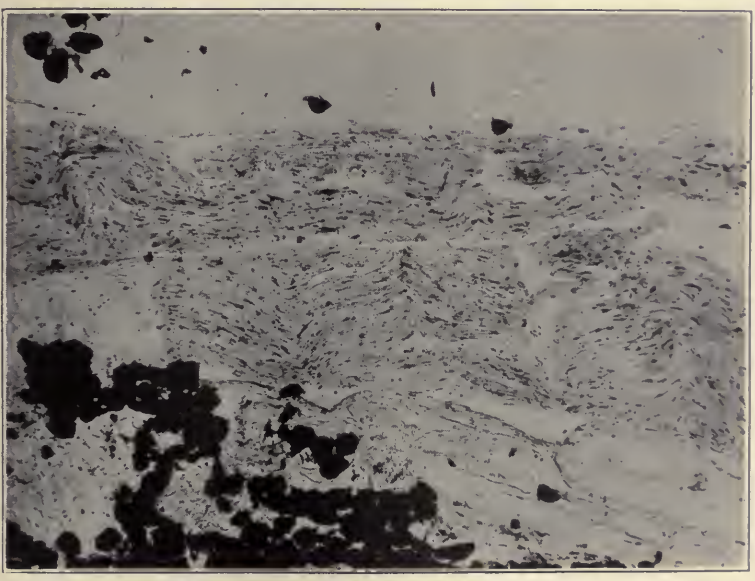

Fig. 9 (Case 4).—Peripheral nerve; Marchi stain.

INCONGRUENCE OF PATHOLOGIC AND CLINICAL FINDINGS

A similar lack of parallelism at times appears between the extent of the pathologic changes and the severity of the symptoms. Minnich and Nonne offer, as a partial explanation of this disharmony, the special location of the lesions in the posterior columns.

One of our patients (U. H. No. 12082), with a severe anemia, paresthesia and absence of vibration sense in the toes and marked impairment in the malleoli, showed no definite findings in the cord; the peripheral nerves, however, were distinctly involved. In another patient (U. H. No. 13141) with severe anemia, some numbness and tingling in the feet and with vibration sense lost in the toes and much impaired in the malleoli, there was no change in the cord except a possible pallor along the dorsal septum, but there was marked degeneration of the peripheral nerves.

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[34] Siemerling, E.: Rückenmarkserkrankungen und Psychose bei perniciöser Anämie, Arch. f. Psychiat. **45**:567-594, 1909.

OPTIC NEURITIS

Optic neuritis is referred to by many writers, but either is not frequently sought or is not often present. It occurred only once in fifty-eight of Collier's cases, and even in this instance was probably of syphilitic origin. However, von Noorden and others have reported eyeground change of some sort as occurring frequently in severe anemias. Additional cases of optic neuritis are reported by Boedeker and Juliusberger,[15] Russell,[35] Hawthorne,[36] Bramwell [37] and Woltmann.[28] Bielschowsky found changes in the optic nerve in two cases. Schroeder found hemorrhage near the macula lutea in one case and Bastianelli [17] found optic nerve atrophy, confirmed at necropsy, in a case of pernicious anemia with spinal sclerosis; in the same case the posterior roots were intact. Putnam and Taylor [23] found optic atrophy in two and probably three out of fifty cases.

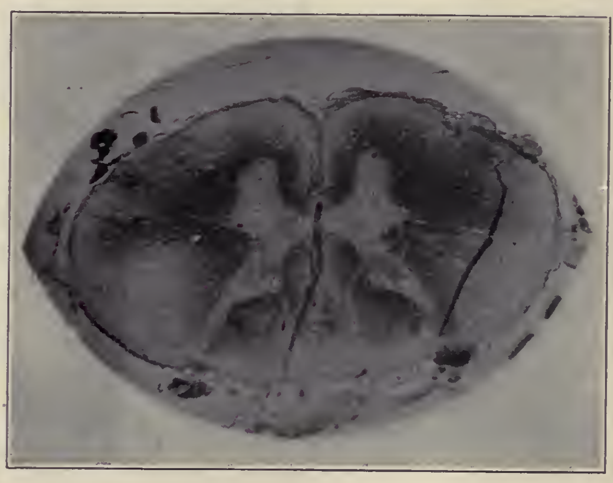

Fig. 10 (Case 5).—Eleventh thoracic segment cord; Weigert stain.

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[35] Russell, R.: The Relationship of Some Forms of Combined Degeneration of the Spinal Cord to One Another and to Anemia, Lancet **2**:4, 1898.

[36] Hawthorne, C. O.: An Address on the Cerebral and Ocular Complications of Anemia and the Probable Relationship of these to Thrombosis, Lancet **2**:857-863, 1908.

[37] Bramwell, Byron: Remarks on a Case of Subacute Combined Degeneration of the Spinal Cord Simulating Disseminated Sclerosis, Brit. M. J. **6**:1396, 1910.

THE PERIPHERAL NERVES

In most of the cases reported it is evident that the peripheral nerves have been studied either insufficiently or not at all. When studied the results have varied greatly.

Lichtheim [5] reported the peripheral nerves intact.

Von Noorden,[38] in 1892, stated that the alterations resembled those of a multiple neuritis with carcinoma. In his case, the crural nerve showed no definite changes but both tibials were markedly degenerated, two-fifths to three-fourths of the nerve fibers showing changes.

Nonne [13] particularly notes that the posterior roots were practically intact. In three of his cases the peripheral nerves were studied and showed no noteworthy degeneration.

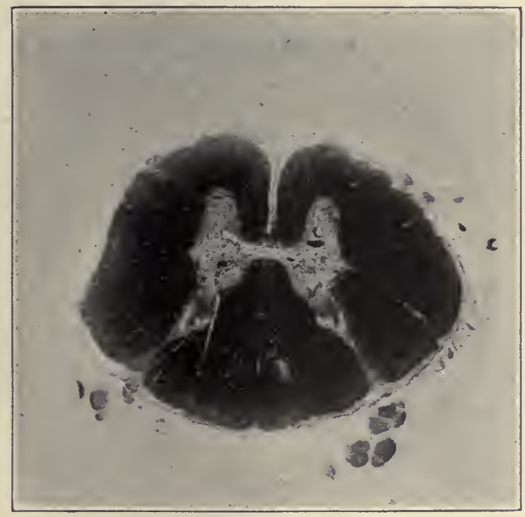

Fig. 11 (Case 7).—Eleventh thoracic segment cord; Weigert stain.

Burr [14] reported in one case slight degeneration in the posterior roots, but Lissauer's zone and the peripheral nerves were intact. Bowman,[39] Billings,[21] Hughes and Spiller [40] found Lissauer's zone and the posterior roots intact, Rothmann [41] found slight degeneration in places in the posterior roots. Boedeker and Juliusberger [15] found the extra medullary roots and the peripheral nerves intact though there was some degeneration of the root entrance zone. Lloyd [16] found some changes in the nerve trunks, the fibers not staining evenly with Weigert's method.

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[38] Von Noorden, Carl: Untersuchung über schwere Anämie, Charité Annalen, 1892, p. 217.

[39] Bowman, H. M.: On the Association of Diseases of the Spinal Cord with Pernicious Anemia, Brain **17**:198-213, 1894.

[40] Hughes, W. E. and Spiller, W. G.: Report of a Case of Pernicious Anemia with Changes in the Spinal Cord, J. Nerv. & Ment. Dis. **28**:573-574, 1901.

[41] Rothmann, M.: Die primären combinierten Strangerkrankungen des Rückenmarks, Deutsch. Ztschr. f. Nervenkrankh. **7**:171-262, 1895.

Russell, Batten and Collier [20] found that usually Lissauer's tract appears normal. Various peripheral nerves were examined and, though usually normal, occasionally slight changes were found with the Marchi or Weigert-Pal method. In one case, however, there was considerable degeneration in both the larger and smaller nerve fibers, interpreted as of late origin.

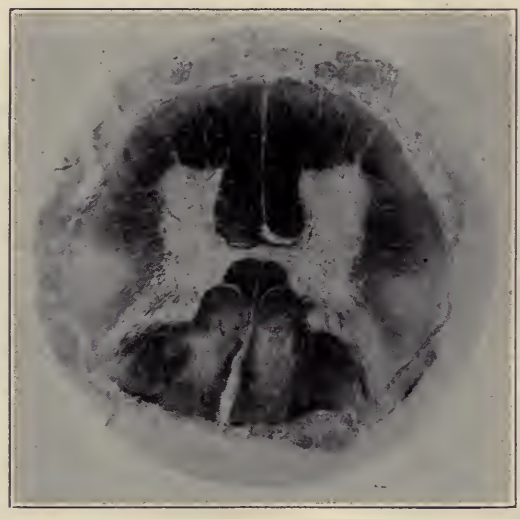

Fig. 12 (Case 8).—First lumbar segment cord; Weigert stain.

Putnam and Taylor [23] studied pathologically five cases, in all of which the patients had distinct sensory disturbances. In all there were relatively insignificant changes in the posterior roots, which the authors interpreted as local nutritive disturbances. Brown, Langdon and Wolfstein [42] stated that "probably Lissauer's zone is intact. In the dorsal and cervical levels the entering posterior root bundles appear slightly involved." Homen [43] found the posterior roots usually free though slight changes may appear with the Marchi stain. In the peripheral nerves few or no changes were found.

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[42] Brown, M. A.; Langdon, F. W., and Wolfstein, D. I.: Combined Sclerosis of the Lichtheim-Putnam-Dana Type Accompanying Pernicious Anemia, J. A. M. A. **36**:552-554, 1901.

[43] Homen, A.: Des lésions non tabétiques des cordons postérieurs de la moëlle epiniëre, Rev. neurol. n. s. **8**:930-941, 1900.

Henneberg[44] (Lewandowsky) believed that the anterior and posterior roots are intact. The numerous black dots at the root entrance zone, exceptionally seen in Marchi preparations, are looked on as postmortem alterations. Bramwell[24] believed absence of lesions in the peripheral nerves to be characteristic.

In our own cases, we have tried to distinguish clearly between changes in proximal nerve trunks and in more distal nerves, but we have avoided small nerves of the extreme periphery on account of the difficulty in determining the presence of changes. As far as possible, we have drawn our conclusions from observations on the anterior tibial studied with the Weigert, Marchi and Bielschowsky methods. We believe that degeneration in the true peripheral nerves may be apparent with the Marchi method when Weigert preparations show nothing, and that the Marchi changes are not of agonal or postmortem origin. The peripheral nerves were studied in seven of the ten cases reported at some length. The Marchi method showed definite degeneration in Cases 1, 2, 3, 4, 8 and 9. The Weigert method gave similar results in Cases 2, 3, 4 and 6, and in Case 1 the nerves stained somewhat irregularly, which may be interpreted as possible evidence of beginning degen-

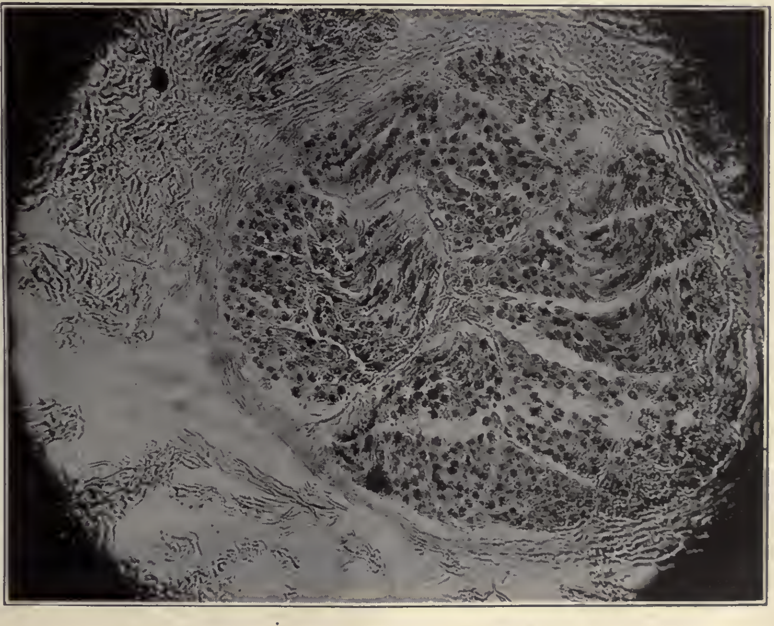

Fig. 13 (Case 8).—Peripheral nerve; Marchi stain.

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[44] Lewandowsky: Handbuch der Neurologie, Berlin, Spezielle neurologie 2, **3**:99, 1910.

eration. A Marchi stain was not made in Case 6, and no peripheral nerves were obtained in Cases 5, 7 and 10.

In two of these cases Lissauer's tract was extensively involved; in two cases the root entrance zone was definitely involved on one or both sides, and in two other cases the degeneration was less distinct but fairly definitely shown by the Weigert method.

RELATION OF SPLENECTOMY AND OTHER FORMS OF TREATMENT AND OF REMISSIONS IN THE BLOOD PICTURE TO THE NEUROLOGIC PHENOMENA

Russell, Batten and Collier [20] note "the absence of any improvement of the nervous symptoms under treatment. From the first appearance of the disease to the fatal event, the tendency was for the symptoms to increase, sometimes halting, sometimes progressing rapidly, but never showing any remission." This is the experience of McPhedran,[30] Decastello [45] and Bloomfield except that the last author noted slight improvement in one case.

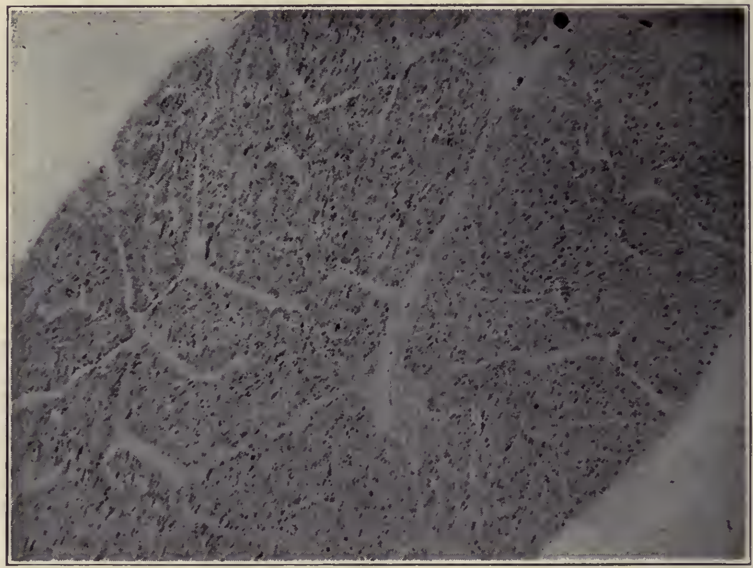

Fig. 14 (Case 8).—Posterior roots; Marchi stain.

Three of our own patients had splenectomy performed. In one, following the operation, there was distinct advance in all the neurologic signs, including increase in the extent of the paresthesias and touch, joint and vibration blunting. The other two patients showed transient improvement both of blood and sensory disturbances.

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[45] Decastello, v. A.: Perniciöse Anämie mit tabeformer Rückenmarksveränderungen und diffuse Sklerodermie, Ztschr. f. d. ges. Neurol. u. Psychiat. **5**:566, 1914.

Though sensory changes in our cases have usually been little altered during remissions in the blood condition we cannot agree that the nervous symptoms never improve. In one case, not reported in our present series, a physician showed marked sensory changes, both subjective and objective, for eighteen months before there was a distinctive blood picture of pernicious anemia. Subsequently he became so ill that he was confined to his bed for several months with loss of patellar and Achilles' reflexes and practically total loss of touch, pain, temperature, joint and vibration sensibility in the feet and legs and considerably less marked changes in the hands. Later his condition improved, and he returned to practice for a year. During this time his blood became almost normal, as did his appreciation of touch and pain stimuli with definite improvement in vibration sensibility and sense of position. After about twelve months the condition of the blood became much worse, his sensory disturbances reappeared, and he died within a short time. Though this is the most striking case in our experience of a remission in the sensory symptoms, in several other cases there was at times a distinct betterment in the course of the disease, capable of demonstration by objective methods. If our contention in reference to the occurrence of peripheral nerve changes be true, it is to be expected that there would be periods of improvement in the sensory findings in some cases.

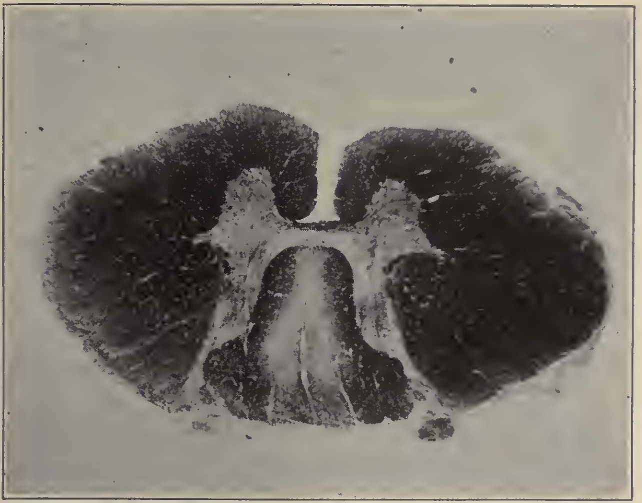

Fig. 15 (Case 9).—Second thoracic segment cord; Weigert stain.

PHYSIOLOGY OF SENSATION AND CORRELATION CF SENSORY AND ANATOMIC FINDINGS

As a basis for possible correlation of the anatomic and sensory findings, we briefly summarize the present views on the sensory tracts of the cord.

Two paths are open to touch: one through the uncrossed exogenous fibers of the posterior columns, another through the opposite anterior segment of the ascending anterolateral column. Impulses arising from painful and thermal stimuli enter the cord in short root fibers and are transmitted to the optic thalamus by fibers of the posterior segment of the ascending anterolateral column (tractus spinothalamic et tectalis).

Fibers for the sense of position and movement (Head and Thompson), vibration (Egger), stereognosis, tactile discrimination (compass test) and appreciation of weight (Head and Holmes, Déjerine, Page May, Sherrington) pass by way of the homolateral dorsal columns.

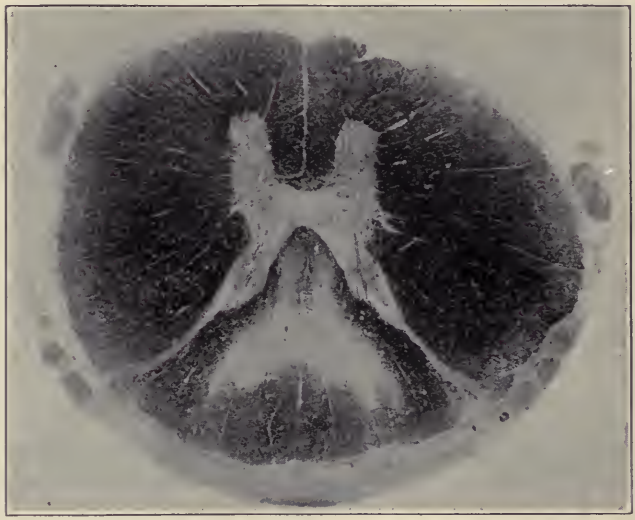

Fig. 16 (Case 9).—Seventh thoracic segment cord; Weigert stain.

Deep unconscious sensibility (afferent impulses underlying muscle coordination and reflex muscular tone) is transmitted to the cord by short and median root fibers, receives collateral stimuli from long root fibers (Déjerine), and passes homolaterally in Gowers' tract and hereterolaterally in the direct spinocerebellar tract.

Déjerine states that "as to deep conscious sensibility (vibration, stereognostic and weight sensations, bone sensibility and tactile discrimination) it follows the course of the long root fibers of the posterior columns of the cord and enters its secondary pathway only at the level of the neck of the medulla."

Petren puts forth the theory that there is a double pathway in the cord for the sense of passive position; one in the posterior column, the other in the homolateral dorsal cerebellar tract. He believes that one of these pathways may be abolished without marked disturbance of passive position sense. From the clinical and pathologic study of a case, Thompson concludes that a complete lesion of the long fibers of the posterior column of the spinal cord results in a loss of the sense of passive position, ataxia, and loss of discrimination of points and weights. Localization of touch is little affected. This result agrees with the experiments of Page May. Painful, thermal, and tactile impulses can ascend the cord, although the posterior columns are degenerated. The evidence derived from Thompson's case is at variance with the view of Petren that a lesion of the posterior column does not produce loss of passive position sense unless the dorsal spinocerebellar tract in the same half of the spinal cord is likewise affected.

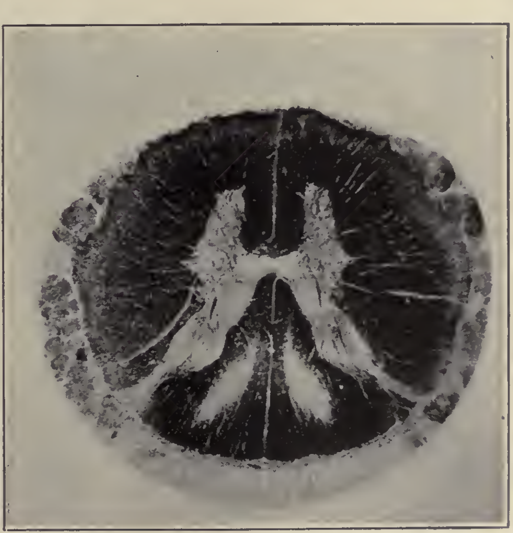

Fig. 17 (Case 9).—Twelfth thoracic segment cord; Weigert stain.

A comparison of the objective sensory with the postmortem findings in ten selected cases of our series gives these results:

Case 1 (J. B.).—Vibration sense was impaired in the fingers and was absent in the feet and ankles. Position sense was normal. Sense of touch was diminished in the tips of the fingers. Superficial pain sense was normal. The cord showed a slight lateral change at the periphery and a slight change along the dorsal groove. There was some degeneration of the anterior and posterior roots and involvement of the peripheral nerves.

Case 2 (S. H.).—Vibration was impaired in the hands and absent in the feet and legs. Position sense was not noted. Sense of touch was diminished in the feet, legs and hands. Sense of pain was much impaired in the feet and legs and less so in the hands. The cord showed marked degeneration in the posterior and lateral columns. Lissauer's zone was much involved, and there was extreme degeneration of the peripheral nerves (Figs. 1, 2, 3, 4).

Case 3 (M. P.).—Vibration sense was absent in the feet and legs. Touch, superficial pain and position sense were all normal. There was marked change in the posterior and lateral columns, the lesions in the posterior columns being mostly "patchy." There was marked degeneration, shown by the Marchi method, of the posterior and anterior roots and the peripheral nerves (Figs. 5, 6, 7, 8).

Case 4 (A. H.).—Vibration sense was absent in the feet. Sense of touch, pain and position were normal. The only change in the cord was a possible pallor along the dorsal groove. The peripheral nerves showed marked degeneration (Fig. 9).

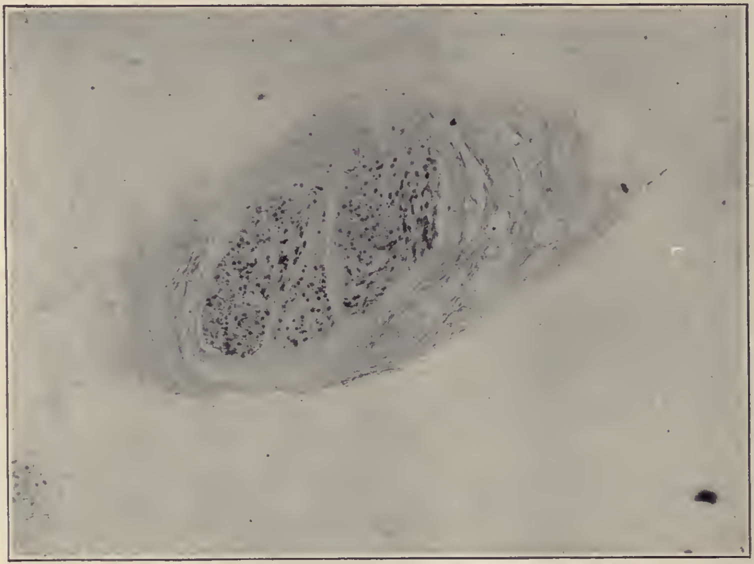

Fig. 18 (Case 9).—Peripheral nerve; Weigert stain.

Case 5 (W. M.).—Vibration sense was not recorded. Position sense was absent in the toes. There was slight diminution of touch and pain sensation in the feet. There was marked change in the anterior, posterior and lateral columns of the cord. Some degeneration of the root entrance zone had occurred. The peripheral nerves were not available (Fig. 10).

Case 6 (A. R. S.).—Vibration sense was not tested. Position sense was absent in the feet and diminished in the hands. Touch and superficial pain sensation were slightly impaired in the feet and hands. The cord showed old sclerosis in Goll's column with patchy lesions in Burdach's column; there were both old and recent degeneration in the lateral columns and definite degeneration of the peripheral nerves.

Case 7 (H. W. L.).—Vibration sense was absent in the feet and legs. There was slight impairment of position sense in the toes. Anesthesia and analgesia of feet, hypesthesia and hypalgesia of the hands and legs were present. There was a sclerotic patch in the middle of one of the posterior columns; a slight change had occurred in the other posterior column; there were practically no lateral changes. The peripheral nerves were not available (Fig. 11).

Case 8 (N. H.).—Vibration sense was absent below the midthoracic region and absent or much impaired in the hands. Position sense was absent in the toes. Sensation of touch was impaired in the feet. Pain sense was normal. There was degeneration in both lateral and posterior columns. The sciatic nerve showed some degeneration (Figs. 12, 13, 14).

Case 9 (H. S.).—Vibration sense was not recorded. Position sense was absent in the feet. There was slight hypesthesia in the toes. The cord showed a slight change in the lateral columns, at the extreme margin, with circumscribed sclerosis in the posterior columns. Degeneration was evident in the anterior and posterior roots and in the peripheral nerves (Figs. 15, 16, 17, 18, 19).

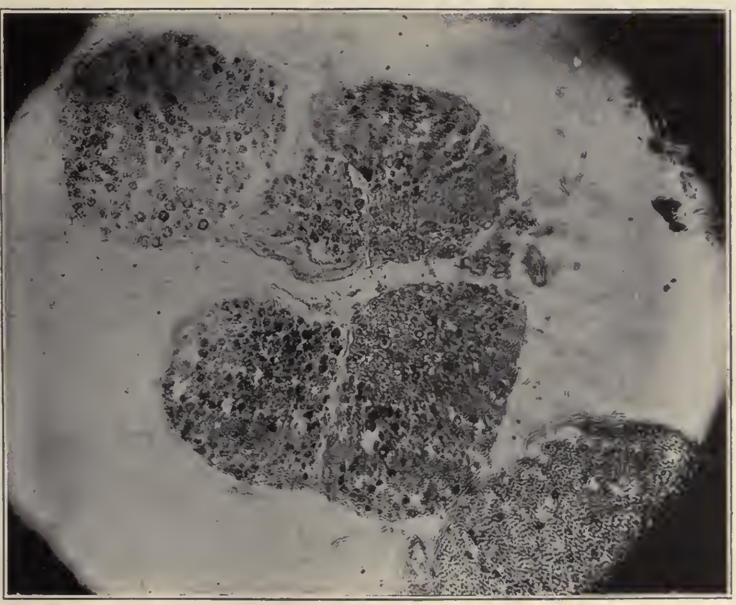

Fig. 19 (Case 9).—Posterior roots; Marchi stain.

Case 10 (A. P. M.).—Vibration sense was absent below the twelfth thoracic segment and much impaired in the hands and arms. Position sense was absent in the fingers, toes and feet. Touch and superficial pain sense were normal. There were a few ballooned fibers in the lateral columns and considerably more, rather diffusely scattered, in the posterior columns with an old sclerotic patch in the middle of each posterior column. The roots were uncertain. The peripheral nerves were not available.

Vibration sense was absent in seven of these cases, and three times it was not recorded. Of these seven, the posterior columns showed slight sclerosis in three, marked sclerosis in three and practically none in one. Of those showing slight sclerosis in the posterior columns, the lateral columns were normal in one and slightly involved in two. In the three patients with marked posterior sclerosis, the lateral columns were similarly affected. The cord with practically no lesions in the posterior columns showed no change in the lateral columns.

In each instance except one, in which vibration sense was impaired or lost, the changes in the cord afforded a more or less adequate explanation in keeping with Déjerine's view. In this one instance a marked peripheral neuritis was present, subjective sensory phenomena were abundant and most unpleasant, and there was impairment of touch sensibility in the fingers, but pain and joint sensibility remained normal, and it is difficult to explain the extreme and selective loss of sense of vibration on the ground of peripheral nerve disturbances alone. In one instance (Case 7, Fig. 11) with marked loss of vibration in feet and legs there was a small clearly circumscribed patch in one posterior column, a less marked change in the other posterior column and practically no lateral change. It may be assumed in this case that the lesion in the posterior columns corresponds to the point of passage of the fibers conducting vibration sensibility.

Position sense was absent in five cases, impaired in one, normal in three and not recorded in one. In all of the five cases, there was marked degeneration of the posterior columns and in three of these the lateral columns were also much degenerated. In the remaining two the lateral columns were only slightly involved, and it would seem that, if joint sensibility were partly carried in the lateral columns, it would not have been wholly lost, as was true in these two cases. In the one case with impaired position sense, the posterior columns showed slight changes with slight lateral changes. Of the three cases in which position sense was intact, in one case the posterior columns showed numerous patches of sclerosis with more or less normal tissue between and marked change in the lateral columns (Figs. 5, 6, 7). The remaining two showed little change in the lateral columns and slight diffuse change in the posterior columns. In the one case, then, with impaired joint sensibility and in two of the three with normal sensibility the condition in the posterior and lateral columns was such as to indicate only a partial loss of function; these cases therefore do not throw any satisfactory light on the problem of the point of passage of the fibers carrying sense of passive position.

Superficial pain sense was absent in one patient who had a sclerotic patch in the middle of one posterior column and slight changes in the opposite posterior column and in the lateral columns. No peripheral nerves were available. Pain sense was markedly involved in one case with extensive sclerosis in the lateral and posterior columns. The two cases with slight hypalgesia of the feet both showed marked sclerosis of the lateral columns as well as of the posterior columns. In the four cases with no impairment of superficial pain sense the posterior and lateral columns were normal in one instance. In a second instance there was slight change in the lateral columns with moderate involvement of the posterior columns and in the other two both posterior and lateral columns were more or less markedly degenerated.

Cotton touch was lost in one case showing only slight change in the lateral columns and in one posterior column and a small patch in the opposite posterior column. It was impaired in three cases of which one showed slight change in the lateral columns and along the dorsal groove and two showed marked degeneration in the lateral and dorsal columns. There was slight impairment in three cases, two of which had marked sclerosis in the lateral and posterior columns, and one had slight degeneration at the extreme lateral margin and a small patch in the posterior columns. Three cases had no involvement of touch sense, the cord being practically normal in one case, the lateral columns showing slight involvement and the posterior showing moderate sclerosis in the second, and in the third both lateral and posterior columns were extensively involved.

CLINICAL ANALYSIS

The following clinical analysis is based on the study of forty-one cases. As the completeness of the study varied in different instances, the tables do not always cover the full number of cases.

TABLE 1.—Clinical Phenomena

Age at Onset Years	Cases
26-30	1
31-35	3
36-40	6
41-45	7
46-50	11
51-55	4
56-60	2
61-65	4
66-70	2
71-75	1

Past Illness.—Under this heading nothing significant was ascertained.

TABLE 2.—Duration of Illness

	Cases
6 months or less	3
7 months to 1 year	12
2 years or less	12
3 years or less	6
4 years or less	2
5 years or less	1
6 years or less	1

Early Symptoms.—The histories were taken by various interns, and although they may have erred in not obtaining all the early symptoms, they were at least free from any preliminary bias.

TABLE 3.—Early Symptoms

	Cases
General weakness	11
Paresthesia or numbness	12
Paresthesia or numbness and general weakness combined	14
Irritative signs, such as pain and soreness	8
Sharp pain	5

It has been our experience that many patients do not distinguish clearly between motor and sensory phenomena in using the term weakness or numbness and on this account the statistics appearing in the foregoing are not altogether reliable.

Complaint on Admission to Hospital.—This was more or less similar to the early symptoms.

TABLE 4.—Symptoms on Admission to Hospital

	Cases
Weakness	12
Paresthesia or numbness	3
Paresthesia or numbness and weakness	27
Irritative signs	10
Constipation	6
Urinary incontinence	6
Bowel incontinence	1
Shortness of breath	9
Pain in abdomen	7

Irritative phenomena differed. One patient complained of tenderness on pressure over both hands; another, of aching pains. One patient complained of pains in the lower extremities and of a sensation of cold in both upper and lower extremities, and one of rheumatism; one patient complained of continuous tinnitus over a period of two years and one of noises in the head. Another spoke of cramps and pains in the legs and feet.

Subjective Sensation.—The most common complaint was tingling and numbness. This was confined to the hands twice and the feet once, and was present in both hands and feet nineteen times. Pain was complained of in the hands and legs five times, in the arms and legs twice and in the arms alone not at all. A band sensation was described twice. Many times the patient found great difficulty in describing the sensation. One physician in particular, who was under observation for over three years, often referred to his sensations and would generally end by saying that it was a queer feeling which he could not accurately describe. One patient spoke of a feeling of "fine gravel in the hands and feet which she would like to pick off." Another spoke of "creepy, crawling feelings" and at times of a "sensation like sand." Another time she said her hands did not "feel right" and in further explanation added: "I can't describe that feeling—a week ago it was different, it was feverish and not stiff like now."

A third patient spoke of "a sort of roughness that came on as weakness." Another spoke of "a roughness—a bad feeling as if the muscles and nerves were wires, as if they were dry." A feeling like electricity passing through the body or hands and arms was often complained of. A sensation like needles so that it was painful to pick up objects was frequent. Some mentioned a wooden feeling in the fingers. One said his fingers felt as if they were made of glass, and that when he tried to pick up something he would be unable to hold it because he could not feel the object in his grasp. One man said it was difficult to tie his shoe strings because his "fingers did not seem to know where they were," and still another spoke of the difficulty or impossibility of buttoning his clothes because of the "sticking" feeling in his fingers.

A patient recently treated (W. G.) had had numbness in his hands and feet for twenty-two months. At the present time his fingers feel as if "they were crusted due to a burn with hot sand." When he touches anything with his fingers or toes a feeling of electricity seems to go through his body.

Objective Sensation.—Most of the sensory tests were made with a wisp of cotton, a pin with a sharp point and a spherical head and a tuning fork (256C). Deep pain sensibility and joint sensibility were also tested.

TABLE 5.—Cotton Touch

	Cases
Hypesthesia of hands and arms	6
Hypesthesia of feet and legs	4
Hypesthesia of arms and legs	10
Anesthesia of hands and arms	2
Anesthesia of feet and legs	6
Normal to cotton touch	10
Not tested	5

Hyperesthesia was present in four instances. Two of the patients suffered greatly from spontaneous pain, the other two did not. One patient showed hypesthesia from the second intercostal space to the lower costal margin. Sensation was normal in the abdomen and hypesthesia was present in the legs. Such diminution of sensation as was present varied from the slightest changes which were distinguished with great difficulty to those in which absolute anesthesia was present. These higher grades of diminution, however, did not go above the fingers or toes except in one instance in which there was practically no sensation up to the twelfth thoracic segment.

TABLE 6.—Pin Prick

	Cases
Hypalgesia of hands and arms	3
Hypalgesia of feet and legs	3
Hypalgesia of both	13
Analgesia of toes	1
Hyperalgesia of hands and feet	4
Hyperalgesia of varying degrees of fifth cranial nerve	7
No loss of sensibility to pin prick	10
Not carefully tested	9

Hypalgesia was present from the second intercostal space to the lower margin of the ribs in one case, as was true of the hypesthesia but, unlike the latter, it did not reappear in the legs. One patient spoke of a "burning sensation lasting several seconds" after being tested with the pin point.

Like hypesthesia, hypalgesia was usually present only in the fingers and toes, but in a fair number of instances it was present in the hands and feet as well, and in a few instances the patient would repeatedly miscall sharp and dull over any part of the extremities.

TABLE 7.—Vibration Sensibility

	Cases
Diminished in upper extremities	9
Diminished in lower extremities	1
Diminished in both upper and lower extremities	2
Absent in upper extremities only	0
Absent in lower extremities only	20
Absent in both upper and lower extremities	3
Normal in both upper and lower extremities	5
Not examined	8

Unlike the sensibility to touch and pain, vibration sensibility, when impaired, was greatly diminished or lost in practically every case over wide areas. In one instance vibration sensibility was found present throughout several examinations in a man who had been ill ten months. Subjectively, he complained of pain in the lower part of his legs and of numbness extending to the elbows and hips. He remained at the hospital nine months, and in the course of this time his subjective sensory trouble became distinctly worse. In all his examinations there were definite impairment of touch sensibility and slight impairment of pin prick. Position sense was present in the fingers and toes at first, but two months after admission it was lost in the toes.

As our table shows, vibration sensibility was very much more impaired in the lower than in the upper extremities. It was wholly absent twenty times in the lower extremities only, three times in both upper and lower extremities, and in no case in the upper extremities alone. How far upward the loss of vibration sensibility extended we are unable to say with any degree of accuracy. In most of our cases it was absent as far up as the malleoli and many times over the tibia, the patella and even higher up. It is worthy of note that in some cases vibration sensibility was absent in one malleolus and present in the other, as Minnich found in one case in reference to superficial pain sense. Vibration sense was lost, therefore, in the upper or lower extremities or in both in twenty-three cases out of forty-one.

TABLE 8.—Position Sense

	Cases
Sense of position absent in toes	9
Sense of position impaired in toes	9
Sense of position normal in toes	11
Sense of position not tested	12

Judged by our statistics, impairment of the sense of position is by no means so common as loss of vibration sensibility and is hardly more common in our records than diminution or loss of sensibility for touch or pain. We believe that loss of sense of position is less common in the fingers than in the toes. In one case (C) both vibration sense and sense of position were greatly impaired in the toes, whereas both were normal in the fingers. It is rather notable in this case that subjective sensory troubles were confined to the lower extremities. In a second case sense of position was normal in the fingers and slightly impaired in the toes. Vibration sense was absent in the toes, but not noted in the fingers. In this case the sensory trouble appeared in the hands previous to its appearance in the feet. In a third case sense of position was lost in the toes in the course of our observation of the patient, though the vibration sense remained at all times present. This patient had definite subjective sensory trouble in both hands and feet and a hypesthesia and hypalgesia of both.

Astereognosis was noted in three of our cases, but the study was not pursued sufficiently far to enable us to draw any conclusions.

Deep muscle pain sensibility was not carefully studied, but in thirteen instances it was noted as normal in the calves, three times increased in the calves and twice impaired, and twenty-three times it was not examined.

A CLINICAL ANALYSIS OF TEN CASES

We have selected ten cases from our series, in which the neurologic examination was more or less complete, for the study of the relative frequency of the sensory findings. These cases were chosen on account of the completeness of the study and not because of the presence of neurologic findings, though it is probable that the presence of many neurologic phenomena would lead to the more complete study of any given case.

Subjective Sensory Disturbances.—Paresthesias were present in all these cases—in eight they occurred early, either alone or combined with weakness or gastro-intestinal disturbances. With few exceptions, the subjective sensory disturbances were present constantly and in greater or lesser degree.

Objective Sensory Phenomena.—Vibration sense was impaired or absent in the lower extremities alone three times and in both arms and legs five times; there was no impairment in two cases.

Position sense was lost in four of these ten cases; it was impaired in two; slightly diminished in one, and normal in three. It was absent or much impaired in four cases in which vibration sense was also absent, but was normal in three cases and slightly impaired in one case in which vibration sense was lost.

Touch, as tested with cotton, was normal in two cases, slightly impaired in two, and greatly diminished or absent in five cases. Twice there was hyperesthesia.

Superficial pain sense, as tested with a pin, showed no involvement in four patients, slight diminution in one and was much impaired or absent in four cases. In one instance there was definite hyperalgesia.

To summarize: One hundred per cent. of the patients had subjective sensory disturbances, 80 per cent. showed loss of vibration sense over several or more bony prominences, and 70 per cent. showed definite involvement of position sense in the toes. Touch sense was diminished or absent in some areas in 70 per cent. and superficial pain sense in 50 per cent. of the patients.

In one case in which vibration sense was later absent in both feet and hands, it was present during the first part of the patient's stay in the hospital at a time when position sense was completely lost in the toes.

CONCLUSIONS

Changes in the nervous system occur in pernicious anemia in 75 to 80 per cent. of the patients as they present themselves in the ordinary physician's practice.

The frequency of these changes is ordinarily underestimated, due to lack of care in the neurologic examination, or even to lack of any neurologic examination.

Subjective sensory disturbances constitute the earliest and most frequent evidence of involvement of the nervous system.

These, with the objective signs also, are often the earliest evidence of the development of pernicious anemia.

The most characteristic sensory findings are the relatively greater loss of deep as compared with superficial sensibility, but there is by no means normal sensibility to touch, prick and other forms of superficial sensibility.

Almost equally as characteristic as the change in deep sensibility, is the complaint of subjective sensory disturbances. Their constancy, their steady progress and symmetrical location in stocking or glove distributions, the inability of the patient to describe them in definite terms and the great distress without ordinarily actual pain to which they give rise, is rarely if ever found in any other clinical condition.

There is a distinct lack of parallelism between the severity of the blood and nervous phenomena, and either may precede the other by a long period of time. Moreover, marked remissions in the blood picture, either with or without splenectomy, may fail to show a betterment in the sensory disturbances equal to that in the blood, and may show none at all.

A similar disharmony may exist between the sensory changes and the postmortem findings in the cord, so that an essentially normal cord may be found in a person who had shown marked sensory disturbances both subjective and objective.

Contrary to the generally expressed opinion, degenerative changes in the peripheral nerves are common in pernicious anemia and constitute an important part of the pathologic anatomy of that disease.

These changes in the peripheral nerves may serve to explain the disharmony between the sensory phenomena and the changes in the spinal cord existing in certain cases, and in all cases have to do with the production of sensory disturbances; they are especially important in those cases in which marked remissions of sensory phenomena occur.

Vibration sensibility may be lost without loss of sense of passive joint movement and vice versa, and it is possible that the long fibers carrying these two forms of sensibility pass up the cord sufficiently separated so that a beginning lesion in the posterior column may involve either one or the other alone at an early stage or, in accordance with Petren's view, it may be that the fibers subserving joint sensibility, at least, may pass up the cord in two pathways.

MYELITIS AND MYELOMALACIA

A CLINICOPATHOLOGIC STUDY WITH REMARKS ON THE FATE OF GITTER CELLS *

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The term myelitis generally covers a great variety of spinal cord lesions, such as inflammations, degenerations and necrobiotic changes, caused by thrombosis, embolism and endarteritis. The clinical picture may be identical, and a proper clinical diagnosis of the actual condition, whether inflammatory, or degenerative, cannot always be made. In one of the cases recorded here the morbid phenomena were most probably due to a pure inflammatory condition of the spinal cord, myelitis, while the second case with the clinical picture of transverse myelitis was due to an extensive softening of the spinal cord with interesting histologic features.

CLINICAL REPORT (DR. BASSOE)

CASE 1.—*History.*—A Chinese laborer, 50 years of age, unmarried, was admitted to the surgical service of Dr. Carl B. Davis in the Presbyterian hospital, April 1, 1920, on account of urinary retention and weakness of the arms and legs. It was quickly determined that no surgical condition was present, and he was transferred to the neurologic service. Two weeks previously he had developed a cough, and the right leg grew weak but was not painful. The other leg and both arms also grew weaker, and he became unable to pass urine. The previous history, as far as it could be ascertained, was negative except for an attack of gonorrhea a year before, but he denied chancre.

Examination.—On admittance the bladder was distended and catheterization was necessary. The pupils reacted normally but the right one was a little larger than the left. He could not walk but in bed was able to move his extremities in a very ataxic manner. The grip of the left hand was much weaker than that of the right. The knee and ankle reflexes were increased and ankle clonus obtained.

Laboratory Tests on Admittance.—The Wassermann reaction of the blood was negative, and of the spinal fluid weakly positive. The cell count was 16. The Ross-Jones globulin test was positive; Lange's gold test: 0122110000.

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* From the pathology laboratories of Cook County Hospital, Chicago, and Illinois State Psychopathic Institute. A brief report of these cases was made to the Chicago Neurological Society, Nov. 18, 1920.

The leukocyte count was 8,100; blood pressure—systolic, 104; diastolic, 66. The urine was negative, and no cystitis developed; catheterization was not necessary after April 5. There was no fever.

Course of Disease.—April 10: Flexion and extension of the right ankle were fairly strong. Dorsal extension of the left ankle was weak. Flexion and extension of the knees were quite strong. Flexion of the right hip was stronger than that of the left. Heel to knee test showed marked ataxia. The weakness and ataxia of the trunk muscles were relatively more marked than in the legs. The grip of the right hand was fair, of the left weak. The left wrist movement was weaker than the right. The patient could hold his arm up. The flexion of the elbows was relatively strong; extension was weaker, especially on the left side. There was moderate ataxia of the hands. There was stiffness of the neck or cranial nerve palsies; the pupils were equal. Bilateral ankle clonus and probably a bilateral Babinski sign were present. Knee reflexes were increased with prolonged patellar clonus on the left. The cremasteric reflexes were obtained, as well as the right abdominal, which was weak; the left was not obtained. Both wrist reflexes were present; the right elbow reflex was present, the left was absent. There was considerable rigidity of the trunk muscles, slight of the left leg, none of the right leg and arm muscles. Sensation apparently was normal; no tremor. Mentality was good.

April 16 the patient could raise himself half way to a sitting posture by using the arms, but with the hands folded on the chest could raise only the head. Abdominal reflexes were as before. Patellar clonus was not obtained; ankle clonus was present but less prolonged; the planter reflexes were doubtful.

Laboratory Tests on April 14.—The Wassermann reaction of the blood and spinal fluid was negative. The cell count was 9. The Ross-Jones test was weakly positive; Lange gold test: 0122211000.

Course of Disease.—May 7: The left grip was still rather weak. All finger movements, including opposition of thumb to little finger, could be made. The trunk muscles were weak, as before. Dorsal flexion and external and internal rotation of the left foot were apparently nil; of the right foot good. Flexion of the left knee was weak; of the right, fair. Extension of both knees was good. Flexion of the left hip was weak; other hip movements were good. Bilateral ankle clonus and slight left patellar clonus were present. Abdominal reflexes were absent.

The condition gradually improved and on July 25, the following note was made: "The patient walked today without a cane. The left leg is still weak, particularly as to eversion of the foot. Left ankle clonus and increased knee jerk persist. The left grip is stronger, and all movements of both wrists are strong.

The patient left the hospital on August 26 and when seen again, on October 15, only little weakness remained.

Comment.—When this case was reported to the Chicago Neurological Society I suggested it might have been one of infection with the virus of epidemic encephalitis, the cord being chiefly affected. However, the absence of cranial nerve symptoms, except for a transient inequality of the pupils and the early retention of the urine, make it more probable that we were dealing with a case of disseminated myelitis of different etiology.

Case 2.—*History.*—The patient was a married woman, 45 years old, of good family history and with good previous health. She had three healthy children, and there was no reason to suspect syphilitic infection. The husband gave a negative Wassermann reaction. The patient was first seen by me, Sept. 22, 1919. Seven weeks previously she had complained of sore throat and rather severe nosebleed. The legs then became stiff, and after a couple of weeks, she complained of pain in the calves and thighs which was severe at times; she had not been able to walk for about three weeks at the time of my visit. The trouble with the legs was followed by numbness and weakness of the hands and frequent, precipitate micturition. Two weeks previously she had complained of diplopia which lasted for a few days only. She became somewhat dull mentally, and her speech was a little indistinct. There was no headache. She was so clumsy that she had to be fed. Occasional involuntary micturition and defecation had occurred.

Examination.—The patient was well nourished, very sluggish mentally, disoriented as to time and place, with slurred, thick speech. The pupils were equal; the left one reacted very sluggishly to light and in accommodation; the right one reacted better. There was nystagmus on looking to the right. There was no definite cranial nerve paralysis. The tongue was heavily coated, not tremulous. The neck was slightly stiff. There was no paralysis of the arms but coarse, irregular movements of the fingers and arms. Marked ataxia was shown on finger to finger and finger to nose tests. There was complete paralysis of both lower extremities. Wrist and elbow reflexes were normal. Abdominal reflexes were not obtained. Knee reflexes were moderately increased. Bilateral ankle clonus and Babinski sign were obtained. The legs were rather flaccid. Touch and pain senses were lost in both legs almost to the hips, but the upper border was very vague. There was complete loss of sense of passive movement in the toes.

Laboratory Tests.—The Wassermann test of the blood was negative, of the spinal fluid, weakly positive. The cell count was 26. The Ross-Jones globulin test was weakly positive; the Lange gold test rather strongly so (4333211100). There was 80 per cent. of hemoglobin. The red cells numbered 4,240,000; the white cells 12,100. The blood pressure was: systolic, 140; diastolic, 68. The urine was cloudy and contained a moderate number of leukocytes. The temperature was 98, the pulse rate 80.

Course of Disease.—September 24: There was more mental confusion. Left ankle clonus was obtained and the right ankle reflex was slightly increased. The knee jerks were increased without clonus.

September 26: The legs were entirely flaccid. Knee and ankle reflexes were absent. The patient complained of pain and soreness across the chest and back in the midthoracic region.

September 28: Considerable stiffness of the neck and apparent difficulty in turning the eyes to the left were present.

September 29: The leukocytes numbered 9,300. Apparently there was complete loss of motion and sensation below the level of the fifth rib. The plantar reflexes were absent. There was still difficulty in getting the patient to move either eye or both eyes together to the left.

October 3: Lumbar puncture was performed. The Wassermann reaction of the spinal fluid was weakly positive. The cell count was 37. The Ross-Jones test was weakly positive. Lange gold test: 1223211000.

October 8: The leukocytes numbered 16,900. There was constant urinary retention. Bedsores were developing rapidly. The temperature, which was normal on the day of admission, rose to 100 F., Sept. 27 and to 101 F., Oct. 11. From that time on the temperature was usually normal in the morning and up to 101 and sometimes 102 F. in the evening.

October 10: There was increasing difficulty in understanding. When told to touch the nose with the left thumb, she fumbled with the right hand. There was a tendency to euphoria.

October 14: Dr. Phemister made deep incisions through the necrotic area over the sacrum without using an anesthetic, but the patient felt nothing. The hands were more ataxic.

November 7: She was asleep most of the day but was restless at night. Her tongue was coated. She did not have a headache or other pain. She had urinary retention as before. There was no nystagmus except some coarse jerkings on looking to the right. The disks were normal with the exception of a suggestion of temporal pallor in the right eye. There were no cranial nerve palsies. Sensation was lost below the level of the fourth rib.

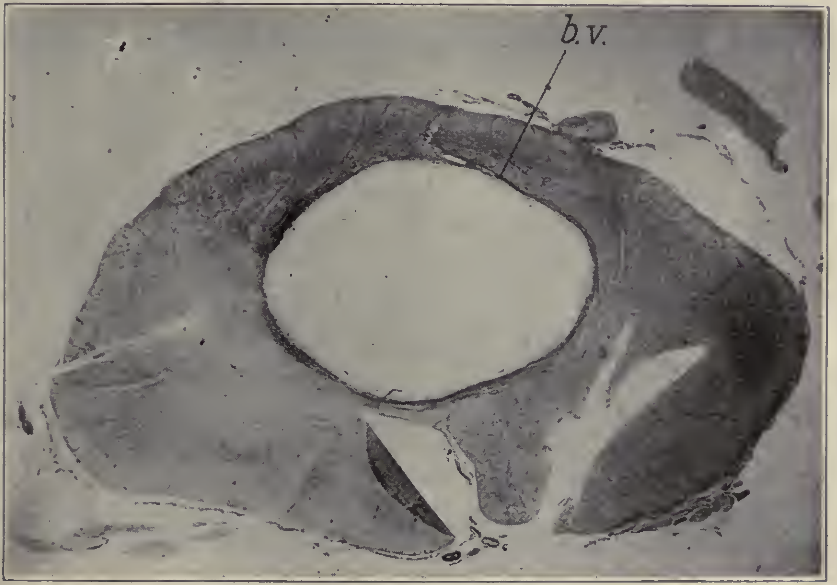

Fig. 1.—Cavity (dorsal region of the spinal cord) lined by a row of gitter cells which in their turn are surrounded by a visible membrane of thickened glia tissue and blood vessels. Alzheimer-Mann stain; × 8.

November 16: Lumbar puncture was performed, which was not felt by the patient. The fluid was clear, the pressure low. The Wassermann reaction of the blood and spinal fluid was negative. The cell count was 19. The Ross-Jones test was weakly positive; the Lange gold test 1222211000.

November 21: The pupils reacted well to light.

December 13: Extensive sloughs were excised from the bedsores over both trochanters.

December 21: The leukocytes numbered 13,600.

During the following month, the irregular fever persisted, the temperature at times reaching 104 F. Paraplegia, anesthesia, retention, bedsores and mental confusion remained as the principal symptoms. The patient gradually failed, and died Jan. 25, 1920.

PATHOLOGIC REPORT (DR. HASSIN)

Macroscopic Changes.—Practically absent in the cerebrum, they were striking in the spinal cord. On section, the entire dorsal portion of the latter was represented by a semiliquid, yellowish, creamy mass escaping freely on the slightest pressure on the membranes, leaving a distinct cavity (Fig. 1). Remnants of anterior and lateral columns could be made out in the upper and middle dorsal regions, while the lower dorsal portion was totally destroyed. Somewhat less damaged was the lumbosacral region in which only the posterior columns were involved. The cervical and lower sacral regions showed only secondary degeneration.

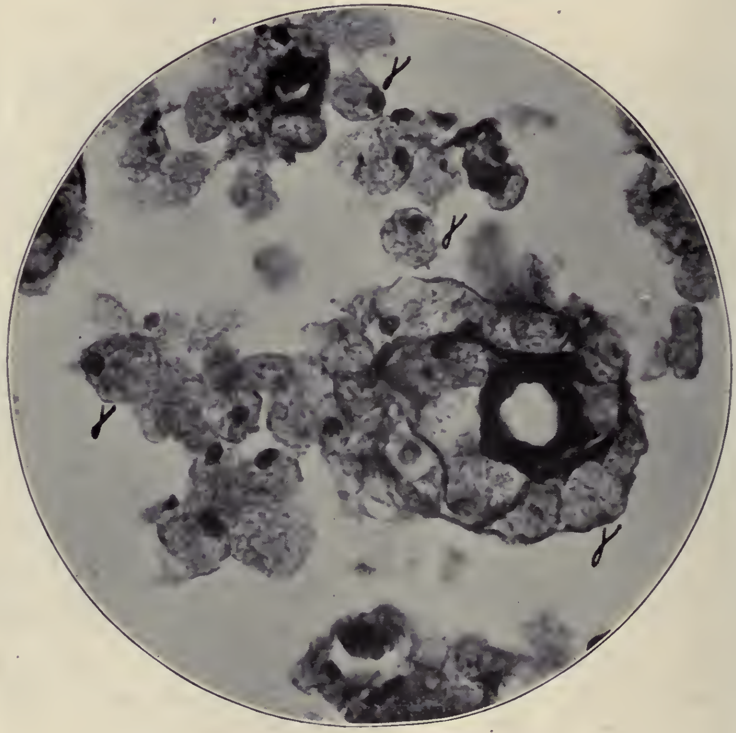

Fig. 2.—Gitter cells γ lying freely and around a small empty vessel enveloped by the ramifications of its thickened adventitia. The freely lying gitter cells show no capsule. Bielschowsky counterstained with Weigert's resorcin fuchsin; × 520.

Microscopic Examination.—Portions of the spinal cord and brain hardened in 10 per cent. formaldehyd have been studied in paraffin and celloidin sections. The dorsal portion consisted mostly of a mass of gitter cells scattered among vessels, broken up nerve fibers and numerous so-called islands of sclerosis. Occasionally one could find preserved ganglion cells. Some segments of the spinal cord showed small patches of gray matter containing practically normal ganglion cells but surrounded by a degenerated, broken up mass of white substance. The remaining gray substance of the thoracic portion appeared histologically normal. Equally normal were the ganglion cells in the less damaged portions of the spinal cord, as well as in the medulla, midbrain, cortical areas and basal ganglions, except the corpus striatum which showed excessive and pronounced neurophagic phenomena.

The better preserved areas of the thoracic region exhibited an abundance of glia nuclei without marked increase in the amount of their cytoplasm. The lumbar region showed numerous gliogenous formations consisting of gitter cells which, in some areas, almost totally replaced the gray substance. The latter could be differentiated from the similarly changed white matter by the occasional presence of practically normal ganglion cells.

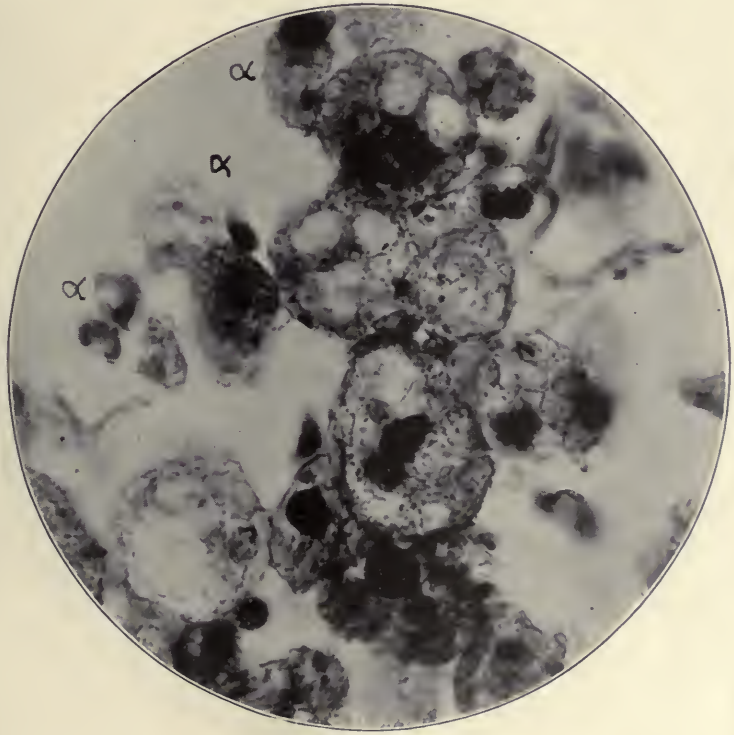

Fig. 3.—Groups of myelophages, large round bodies containing two, three and more large vacuoles with remnants (dark irregular bodies) of nerve tissue within. At α, gitter cells with an eccentric nucleus and a distinct vacuole. Bielschowsky counterstained with resorcin-fuchsin; × 1200.

The blood vessels of the gray matter were numerous, hyperemic, with thickened walls, but without perivascular infiltrations. A few hemorrhages were observed, but without any reactive phenomena on the part of the glia or mesodermic elements. They most likely were terminal manifestations.

White Substance.—Thinly spread between two slides and stained, some with a 1 per cent. solution of methyl-blue-eosin (Mann's mixture), others with osmic acid, scarlet red and Nile blue sulphate, the creamy mass showed an enormous number of various gliogenous formations, principally myelophages and gitter cells. Most numerous were gitter cells of Jacob's γ-variety, in the form of round cell bodies, devoid of processes as well as of a visible membrane, with an eccentric nucleus and a distinct reticular or latticelike structure (Fig. 2). Invariably they were packed with small globules of fat. Certain cells appeared coarsely vacuolated (Fig. 3), with remnants of nerve tissue within some of the vacuoles. They are best classified as myelophages which are phagocytic cells. They usually contain fragments of myelin, which is transformed by them into fat. According to Jacob, the fat is deposited in gliogenous formations generally termed gitter cells. Such cells, of the γ-variety, were freely scattered in the tissues or gathered around the blood vessels, preferably the smaller ones, in the form of concentric rings (Fig. 2). The rings of γ-cells were separated from each other by connective tissue strands, with prolongations surrounding each individual gitter cell. The latter thus appeared as if invested by a connective tissue membrane, a condition never seen in the ordinary gitter cells lying free. These membranes, as well as the strands of connective tissue dividing the rings of the gitter cells, were seen to be connected with the adventitia of the vessel wall.

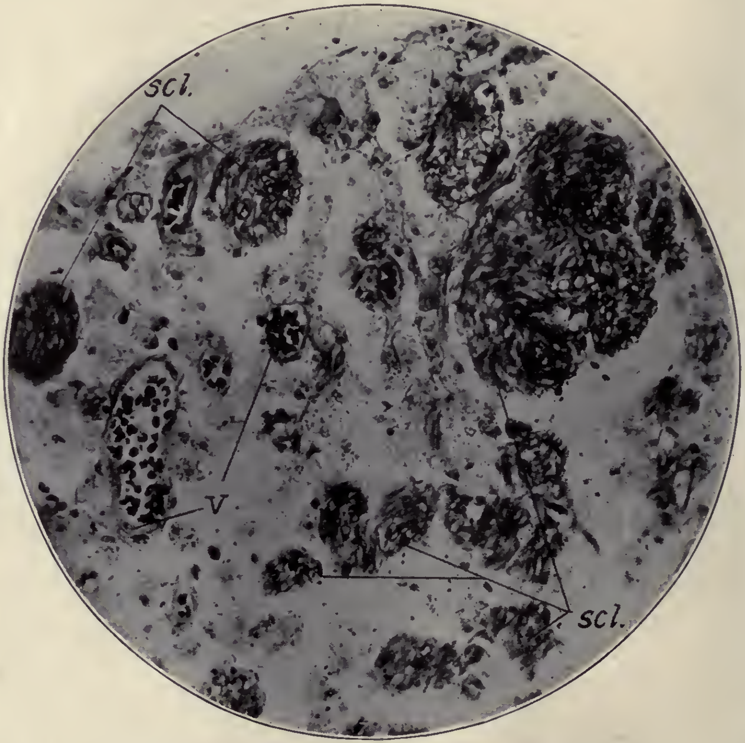

Fig. 4.—Islands of sclerosis, scl; hyperemic small veins, v; the rest of the field is covered with gitter cells and broken up nerve fibers. Paraffin section of an emulsified (softened) area. Bielschowsky stain; × 275.

Striking also was the presence around some capillaries of gitter cells still lying free but ready, so to speak, to be enveloped (Fig. 2) by the proliferating adventitia. It is to be noted that only the so-called γ-variety of gitter cells could be found near or within the adventitia, for only such gitter cells are ready to discharge their fat. Myelophages, gitter cells of other varieties, such as those possessing vacuoles or processes, were not to be found within the adventitia.

Aside from the vessels surrounded by rings of gitter cells, there were a great many vessels densely surrounded, muff-like, by connective tissue which, however, showed no gitter cells in its meshes (Fig. 4). Such vessels had a barely visible lumen, but an enormously thickened wall. In fact, they appeared as islands of connective tissue or sclerosis, greatly resembling the islands in syringomyelia.[1] One gains the impression that they originate from the vessels surrounded by the gitter cells as well as from the connective tissue rings which, after their contents, the gitter cells, have been removed, ultimately shrink forming islands of sclerosis. In many places, the field is represented merely by rows of pale gitter cells surrounding an empty space, or cavity. The rows of gitter cells lining the cavity are remnants of a great mass of similar structures that must have filled the space before the softening had reached the stage of cavity formation. The large cavity shown in Figure 1 was surrounded by a capsule rich in protoplasmic glia cells, blood vessels and glia fibers. Resem-

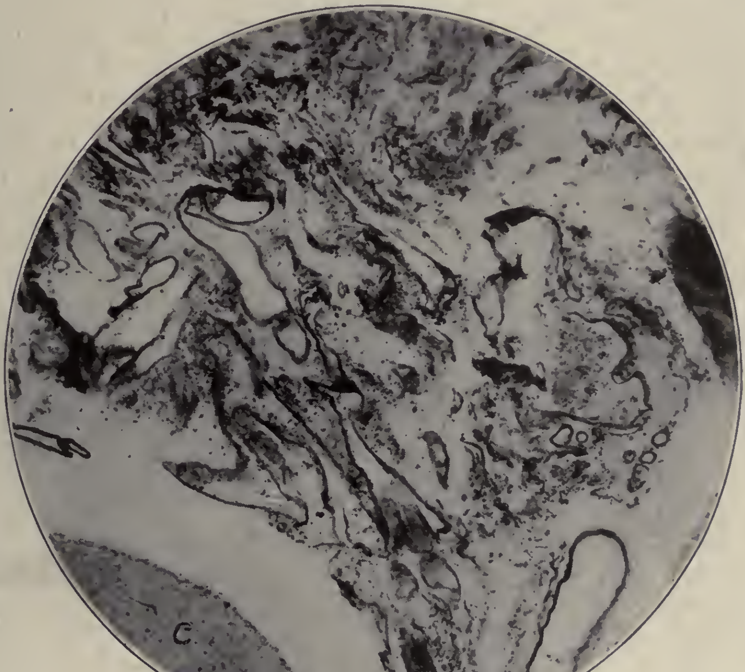

Fig. 5.—The cerebellar pia. Excessive vascularization, hyperplasia and cellular infiltration of the distended meshes; *c*, cerebellum. Van Gieson stain; × 80.

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[1] Hassin, G. B.: A Contribution to the Histopathology and Histogenesis of Syringomyelia, Arch. of Neurol. & Psychiat. **3**:130 (Feb.) 1920.

bling, at first glance, a similar formation commonly seen in syringomyelia, the described cavity is histologically and etiologically entirely different from that of syringomyelia.[1]

Scattered among vessels, vascular islands of sclerosis and gitter cells were nerve fibers, naked axons and fragments of myelin (Fig. 4). The nerve fibers were often practically unchanged, merely broken up into more or less well stained fragments, while some showed an enormously swollen or tumefied myelin sheath which was usually broken up into smaller fragments enclosed within myelophages and gitter cells. In areas that escaped softening, such as Gowers' tract, Flechsig's tract and the anterior colums, the nerve fibers were practically normal. The glia also was preserved, exhibiting an abundance of glia nuclei and so-called protoplasmic glia cells. The latter were often very large, in some instances resembling monster glia cells, and by their homogeneous staining, plump processes and eccentric nuclei, recalling at first glance so-called ameboid glia cells. However, if stained with Alzheimer-Mann mixture, Bielschowsky's method or even toluidin blue, they appeared exceedingly rich in chromatin nuclei and displayed a great wealth of ramifying protoplasmic processes. Nor could we find the "Füllkörperchen" of Alzheimer, or the "Abbau" products so abundantly present in some toxic-infectious conditions. In short, signs of regressive glia changes were totally absent. On the contrary, the glia exhibited marked progressive or proliferative phenomena, not only in the better preserved portions of the spinal cord, but also in those profoundly changed.

The vessels, as already noted, while exceedingly numerous in the destroyed area, mostly in the form of small capillaries with patent lumen and thickened adventitia, were practically normal in the preserved regions of the cord, as well as in the rest of the central nervous system. Only the lower medulla and the region of the brachium pontis exhibited vascular changes. In the medulla, some vessels were thrombosed and surrounded by an area of sclerosed tissue; in the pons there was a quite pronounced perivascular infiltration with plasma cells and lymphocytes.

The pia appeared hyperplastic, thickened and frequently packed with gitter-cells. The pia-arachnoid of the anterior longitudinal sulcus exhibited a great wealth of distended vessels, as well as various hematogenous elements, such as erythrocytes, leukocytes and plasma cells, while at some levels the subarachnoid space contained hemorrhagic foci. At other levels, instead of hemorrhages there were many gitter cells, mostly gathered within the meshes of the pia, around the vessels and, what is rather striking, in the posterior roots themselves.

Equally noteworthy were the findings in the cerebral subarachnoid space, especially at the base, around the optic chiasm, cerebellum and the occipital lobe. Figure 5 shows the pia-arachnoid around the cerebellum, with wide meshes, an enormous amount of blood vessels and various infiltration elements, such as lymphocytes, fibroblasts, clusters of mesothelial cells, gitter cells and hyperplastic strands of the pia-arachnoid. In other cortical regions such infiltrations were less in evidence, but hemorrhagic foci of various dimensions were quite common. However, as in the case of the spinal cord, the hemorrhagic foci were devoid of reactive phenomena and should therefore be considered as terminal phenomena. The choroid plexus was greatly vascularized and showed an abundance of connective tissue. The cells invariably showed the presence of fat in specimens stained with scarlet red and otherwise appeared unusually large and vacuolated.

Summary of Findings.—The findings were:

1. Absence of any inflammatory phenomena in the spinal cord or any other portion of the central nervous system, except some areas of the pons.
2. Vast degeneration of the spinal cord, especially the posterior portion, including some of the posterior roots.
3. An abundance of various gliogenous formations, such as gitter cells and myelophages, which almost totally replaced the degenerated areas.
4. Marked infiltration of distended adventitial meshes, pia and posterior roots with gitter cells and abundance of islands of sclerosis.
5. Hemorrhages in the subarachnoid space of the cervical region of the spinal cord.
6. Cellular infiltration of the distended pial meshes of the brain, especially around the base.
7. Fat infiltration of the choroid plexus.
8. Absence of regressive changes in the glia, in the presence of profound parenchymatous lesions.

DISCUSSION

The histopathologic findings here outlined in a typical case of so-called "yellow" softening of the spinal cord, myelomalacia, are analogous to those found in acute softening of the brain, encephalomalacia. As the latter is no longer considered an inflammatory process, neither can myelomalacia be looked on as a myelitis. Yet, inflammatory conditions ultimately may cause the softening described, for instance, in poliomyelitis, Pott's disease,[2] syphilitic cord lesions, and other conditions.[3] However, the areas of softening in such cases are usually small and circumscribed, while in the case under discussion, they involved the posterior portion of almost the entire spinal cord. The same is practically true for spinal cord softening due to intoxications as experimentally so well proved and studied, among others, by Lotmar.[4]

By far the most frequent cause of softening is vascular thrombosis ("thrombotic softening of the spinal cord" or "spinal thrombosis" of Singer [3]). According to Mager,[5] 80 per cent. of cases of so-called acute myelitis are due to necrobiotic or degenerative processes, usually brought on by vascular changes (infiltrative or proliferative). In the present case such changes were lacking. That the dorsal region was principally affected is probably due to the fact that the blood supply of this region is generally inefficient, and thus more easily implicated. Whatever the actual mechanism or nature of the vascular disorder in this case, it is beyond question that the changes of the spinal cord were similar to those found in secondary degeneration. A typical instance of the latter is represented in this case by the posterior columns of the cervical region. The greatest portion of the nerve fibers were destroyed and replaced by numerous gliogenous formations, such as protoplasmic glia cells, abundance of glia fibers, an enormous amount of gitter cells and glia nuclei. All these formations were kept together, preserving the shape of the posterior column, while in the region of the softened area, the latter was a shapeless mass. In short, the gitter cells harboring the nerve tissue transformed into lipoid substances evidently tend to desert the destroyed area, to become "absorbed." The question arises: How do they accomplish the process of absorption, or what is their ultimate fate? Schmaus,[6] Nissl,[7] Merzbacher,[8] Marchand,[9] Held [10] and Jacob [11] tried to solve this problem. For instance, Schmaus and Nissl thought that the fat-laden gitter cells reaching the vessels unload their fat in the adventitial cells. Jacob is more explicit. According to this investigator, the fat taken over by the mesodermic elements of the vessels is "rebuilt," the adventitial cells turning into gitter cells. Merzbacher believes that these cells are absorbed either through the process of undergoing regressive changes and perishing or by being carried away in toto. He could find them, somewhat modified in structure, in the pia which he calls "Ablagerungsort" (dumping grounds). They reach the pia, he thinks, either by way of the lymph spaces or through the transformed adventitial cells. Finally, he admits that he does not know how the landing of the gitter cells in the adventitial spaces is accomplished, as neither he himself nor any one else ever succeeded in following up the process of direct invasion of the lymph spaces.

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[2] Hassin, G. B.: Histopathologic Changes in Five Cases of Myelitis, Med. Rec., 1916, October 7.

[3] Singer, H. D.: The Pathology of So-called Acute Myelitis, Brain **25**:332, 1902.

[4] Lotmar, Fritz: Beiträge zur Histologie der akuten Myelitis und Encephalitis, sowie verwandter Prozesse. Nissl-Alzheimer's Arbeiten **6**:245, 1913.

[5] Mager, W.: Ueber Myelitis acuta, Obersteiner's Arbeiten aus dem Institut für Anatomie und Physiologie an der Wiener Universität **7**:1, 1900.

As shown in part by Figures 2, 3 and 4, the great majority of gitter cells are either freely scattered over the parenchyma of the spinal cord (Fig. 2), or are enclosed within (1) the meshes of the proliferated adventitia of the vessels, (2) the pia, (3) the subarachnoid space, or (4) the posterior roots. Wherever located, they are similar in structure;

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[6] Schmaus, H.: Vorlesungen über die pathologische Anatomie des Rückenmarks, 1901.

[7] Nissl, F.: Kritische Bemerkungen zu H. Schmaus: Vorlesungen über die pathologische Anatomie des Rückenmarks, Centralbl. fur Nervenheilk u. Psychiat. **26**:88, 1903.

[8] Merzbacher, L.: Untersuchungen über die Morphologie und Biologie der Abraumzellen im Zentralnervensystem, Nissl-Alzheimer's Arb. **3**:1, 1910.

[9] Marchand, F.: Untersuchungen über die Herkunft der Körnchenzellen des Zentralnervensystems, Ziegler's Beitr. z. pathol. Anat. **45**:161, 1909.

[10] Held, H.: Ueber die Neuroglia marginalis der menschlichen Grosshirnrinde, Monatschr. f. Psychiat. u. Neurol. **26**:336, 1909.

[11] Jacob, A.: Ueber die feinere Histologie der sekundären Faserdegeneration in der weissen Substanz des Rückenmarks, Nissl-Alzheimer's Arb. **5**:1, 1912.

the gitter cells of the perivascular or adventitial spaces, for instance, being entirely similar to those present in the meshes of the pia. The sole difference, as noted in the foregoing, is that some gitter cells, especially those freely scattered, are devoid of a connective tissue membrane which is so well seen around the cells within the adventitial spaces. These membranes can be seen originating from the adventitia whose ramifications form numerous meshes harboring the fat laden gitter cells (Fig. 2). Once within the meshes of the adventitial membrane, the gitter cells are carried away by the tissue fluids toward the arachnoid space in the same manner as is the case in the brain.[12]

The gitter cells found in such great numbers underneath the pia are removed in a somewhat analogous manner. They are gradually enveloped by the proliferating pia which thus brings them within the domain of the subarachnoid space and the posterior roots. An analogous process on the part of the pia is to be seen in syringomyelia in which the débris of glia and other tissues are actively removed by this proliferating membrane. However, while in syringomyelia the process of removal of destroyed tissues is exclusively carried out by the pia, this is in myelomalacia the final stage in ridding the nervous system of waste elements. The preliminary work is done by the glia which first transforms the damaged nerve tissue into lipoid substances and carries them to the mesodermic tissues, vessels or pia. The latter, by active proliferation envelop these harmful elements, and finally eliminate them to the subarachnoid space, "the dumping ground" of the central nervous system.

CONCLUSIONS

1. Myelomalacia is not an inflammation, but a degeneration of the spinal cord.

2. The degenerative or regressive nerve fiber changes are associated with progressive glia phenomena, principally in the form of gitter cell formation.

3. The gitter cells in this as in any other type of secondary degeneration, are gliogenous formations. Their task is to transform destroyed nerve tissue into fat-like substances and to transport these substances to mesodermic structures, such as blood vessels and the pia.

4. Reaching these structures, they are actively taken up by the proliferating adventitial and pial elements.

5. From the adventitial and pial spaces they are carried to the subarachnoid space and thence to the blood for final elimination.

6. The subarachnoid space is to be looked on as the "dumping ground" for any waste accumulated in the central nervous system.

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[12] Hassin, G. B.: Histopathology of Brain Abscess, Arch. Neurol. & Psychiat. **3**:616 (June) 1920.

EPIDEMIC ENCEPHALITIS

CLINICAL AND PATHOLOGIC STUDY OF TWENTY-FIVE CASES

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INTRODUCTORY

In a previous communication, H. E. Allen and I[1] reported four cases of lethargic encephalitis as seen in the early part of the epidemic, and reviewed the literature and cases reported up to that time. After a short interval, during which few cases were seen in our community, a sudden recrudescence occurred. During this recrudescence I had the opportunity to study twenty-five cases of the disease and also to make necropsy examinations of several. These cases I shall report.

GENERAL CONSIDERATIONS

Age and Sex.—The youngest patient was 16 years of age; the oldest 61. Nineteen of the twenty-five were between 25 and 40 years old. Eighteen were males and seven females.

Relation to Influenza.—Direct: Of the twenty-five patients observed, only two gave a history of influenza. It consisted of a mild attack six weeks and four months, respectively, prior to the encephalitis.

Indirect: A definite relation between epidemic influenza and epidemic encephalitis was observed. The great epidemic of influenza raged during the months of October and November, 1918. We recognized the first case of epidemic encephalitis in February, 1919. Between February and October, 1919, we saw four typical cases, but from that time to February, 1920, hardly any cases were seen in the community.

A recrudescence of the influenza epidemic occurred in January, 1920, and lasted until Feb. 20, 1920, when cases of encephalitis began to appear. They gradually increased in number and reached their height in the latter part of March, when most of the cases were seen. On one day—March 24—five patients with encephalitis were admitted to the Waterbury Hospital.

Mode of Onset and Symptoms.—Premonitory symptoms of lassitude and general malaise of several days' duration were complained of by ten of the patients. The most frequent mode of onset (sixteen cases) was with neuritic pain. In the majority of instances it commenced rather abruptly. Jerking and twitching were frequent symptoms and were among the first complained of in eighteen of the cases. In five cases the early symptoms were suggestive of a mild meningitis: rigidity of the neck, headache, tâche cerebrale and a positive Kernig's sign.

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[1] Alexander, M. E., and Allen, H. E.: Lethargic Encephalitis, Arch. Neurol. & Psychiat. **3**:485 (May) 1920.

Fever: A temperature above 100 F. was found at one time or another in all cases observed. In eighteen cases it was 102 F. or above. It was highest in the cases associated with a great deal of twitching and neuritic pain and in those that showed signs of cortical involvement. With exacerbations the temperature generally rose

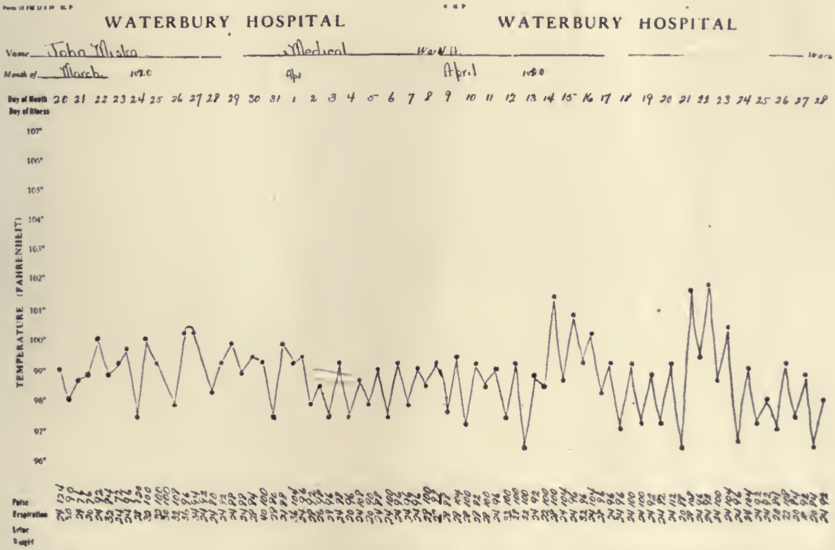

Fig. 1 (Case 10).—Usual temperature elevation in the meningoradicular type of encephalitis and the elevations with recrudescences of the disease.

(Fig. 1). In two cases hyperpyrexia was noted (Fig. 2). Both terminated fatally. The rise of temperature was irregular and unless taken at frequent intervals might have been overlooked in some cases.

Pulse: The pulse presented the usual features of infectious diseases. It averaged between 90 and 100 and was generally a little faster than one would expect with the comparatively slight elevations of temperature. In patients showing evidence of marked disturbance of the basal nuclei (dysphagia and marked lethargy), the pulse was generally between 100 and 110. A rate above 120 was uncommon, and when it persisted was of serious import. Extrasystole (transient) was noted in two cases; other irregularities of the pulse were absent.

Blood Pressure: This was generally slightly elevated, not only during the stage of excitement but even during pronounced lethargy. In one case peculiar, temporary elevations of blood pressure were observed. On admission to the hospital it was: systolic 120; diastolic, 70. On the second day it was systolic, 190; diastolic, 100. Similar fluctuations were noted on two other occasions.

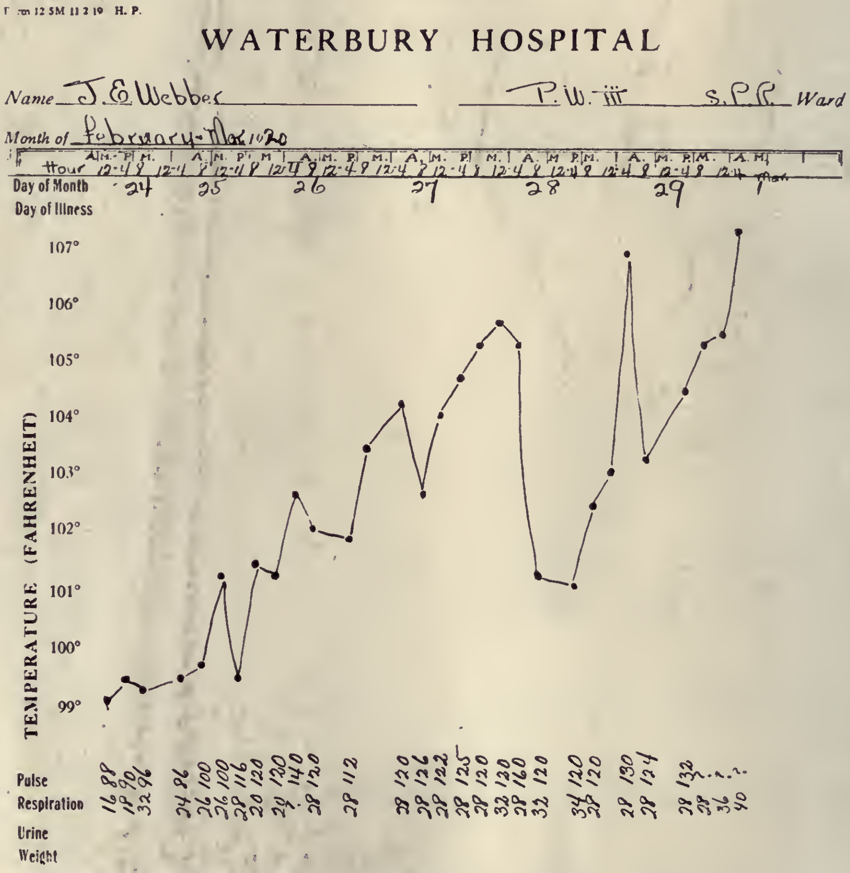

Fig. 2 (Case 16).—Temperature elevation showing hyperpyrexia. Necropsy examination revealed no complications and no pathologic findings except at the base of the brain.

CENTRAL NERVOUS SYSTEM

Restlessness and wakefulness were present in eighteen patients. In five, lethargy set in after a preliminary period of restlessness and insomnia lasting two or three weeks. These cases particularly emphasized the fact that the various cases which at certain times showed symptoms so vastly different, really belonged to the same affection

In most of the cases the lethargy, listlessness and masklike face were not present until the third or fourth week. Several cases, commencing with choreiform twitchings, involving at different times almost all muscles in the body, including the abdominal twitching so characteristic of this group, gradually developed into the lethargic type, with ptosis of one or both lids, diplopia and facial palsies. The patients remained in a state of lethargy and somnolence for several weeks and then, with gradual awakening of the sensorium, the various twitchings reappeared. In two cases the process was repeated three times.

Cranial Nerves.—Involvement of the cranial nerves was not so common in this series as in the earlier epidemic.

First Cranial Nerve: In the first ten cases the olfactory nerve was not examined. After noting small hemorrhages and perivascular infiltration in this nerve in one of the cases examined postmortem, the sense of smell was tested in all patients. It was found markedly diminished in four cases, absent in none.

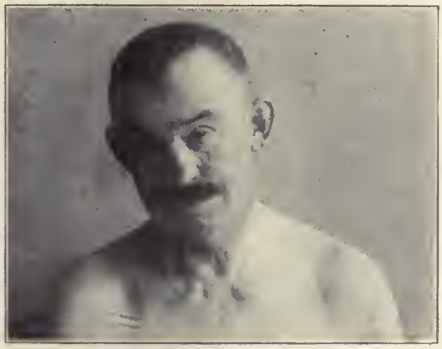

Fig. 3 (Case 8).—Spasm of sternomastoid muscle with torticollis.

Second Cranial Nerve: No disturbances of the optic nerve were noted except in two cases. In these the diameter of the papilla seemed enlarged, the veins moderately engorged and tortuous. Around the papilla the retina was slightly opaque and striated. One of these patients died, and microscopic examination showed evidence of optic neuritis. The other patient recovered and six months after the illness all signs of choked disk had disappeared.

Third Cranial Nerve: This nerve was involved in ten cases; diplopia in four; ptosis in six; strabismus in seven. In many the various paralyses were present at different times, but they were not common at the beginning.

Fourth Cranial Nerve: Paralysis was present in three cases.

Fifth Cranial Nerve: Difficulty in mastication was present in two cases, and the masseter muscles did not seem to contract. Neither patient was lethargic, nor was there pronounced general weakness. In one of these there was hyperalgesia on one side of the face and an abundant crop of herpes along the upper lip. In the other there was diminution of sensibility of the lips and tongue. This patient also had excessive salivation. After six days the anesthesia gradually disappeared.

Sixth Cranial Nerve: Conjugate deviation of the paralytic form was present in four cases.

Involvement of the third, fourth and sixth nerves, as evidenced by total ophthalmoplegia, was present for a short time in two cases.

Seventh Cranial Nerve: Incomplete facial palsy was present in nine cases; in one the paralysis was bilateral.

Eighth Cranial Nerve: Diminution of the acuity of hearing was noted in six cases. Four patients complained of distressing and persistent noises in both ears.

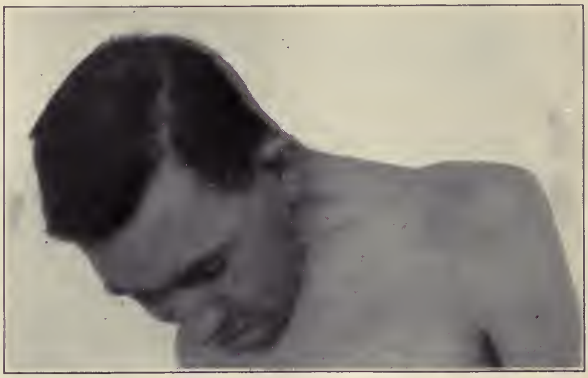

Fig. 4 (Case 11).—Spasm of sternomastoid with torticollis; also paralysis of the muscles of the back of the neck.

Ninth Cranial Nerve: Dysphagia was present in eleven cases, lasting from three days to four weeks. In all cases it was associated with involvement of other cranial nerves, and it was difficult to determine how much the vagus or hypoglossal involvement contributed to the symptom.

Tenth Cranial Nerve: Unilateral flaccid paralysis of the soft palate was noted in two cases. Speech was nasal and deglutition difficult. There was also paralysis of the vocal cord on the same side. Marked gastric irritability and vomiting without apparent cause were present in two cases. Bulimia was present in one case, accompanied by paralysis of the vocal cords and cardiac arrhythmia—extra systole. The bulimia lasted about ten days and gradually disappeared. Irregular respiration was present in one case. It resembled very much the pulse rhythm of a sinus arrhythmia, being mainly trigeminal in character.

Eleventh Cranial Nerve: Persistent spasm of the sternomastoid muscle was seen in one case (Fig. 1). There was paralysis of the trapezius in the same patient and to a still more marked degree in another patient (Fig. 4).

Twelfth Cranial Nerve: Unilateral paralysis of the tongue was noted in one case. Movement of the tongue within the mouth was imperfect on the right side, more so in the attempt to protrude the tongue.

PERIPHERAL NERVES

Pain.—It was the first symptom complained of in sixteen of the cases, most frequently in the face and one side of the neck, next in the extremities. The majority of the patients complained of tenseness and discomfort in the extremities and along the course of various nerves, but at times of sharp and lancinating pains. In one case it was localized in the spine and darted alternately to the right and left. In three patients the lancinating pains were very distressing, frequently sudden and flashing in character and accompanied by mild sensations of contraction. In one patient these pains persisted for four days, requiring morphia to allay them.

Sensory Disturbances.—No dissociation of sensations was observed in eleven of the cases examined for it. Hyperalgesia was noted in three cases. In one it was associated with trophic disturbances, such as blebs and bed sores. No thermo-anesthesia was found.

Trophic and Vasomotor Disturbances.—Persistently cold, clammy and cyanotic finger tips were observed in four patients, profuse sweating in three, excessive salivation in two, decubitus in three.

Tremor.—Coarse tremor was present at some period or other in twenty cases. Its initial location and progress were ascertained in fifteen cases. In six patients it began in one extremity, nearly always a hand, and spread to the lower extremity of the same side; in three it commenced in one side of the face; in two in the abdominal muscles, in one in the muscles of the neck and shoulder, while in the rest it was practically simultaneous in nearly every part of the body.

Slight at first, it became more marked later, so that the affected part would move sufficiently to disarrange the bed clothes. The rate varied. In one patient it was 80 a minute; in others it varied from 20 to 30 a minute. Generally disappearing during sleep, in three cases it persisted, although to a lesser degree. The patient was generally able to diminish or even to suppress it entirely for a few seconds or minutes, when it became entirely uncontrollable. Finer motions and manipulations were not affected very much: and the patients were generally able to feed themselves and write short notes, except during the height of the disease when the jerkings were extremely violent. Kymographic tracings revealed contractions at irregular intervals but generally of about the same amplitude.

Among the more peculiar and extraordinary spasms were those of the abdominal muscles. The upper part of the recti abdominis and external oblique muscles would contract and twitch, varying from 20 to 50 times a minute. At first the spasm resembled the movements of hiccup but observation showed that it had no relation with respiration or with contractions of the diaphragm. It was generally accompanied by simultaneous spasm of the extremities and in some instances of nearly the entire voluntary body musculature.

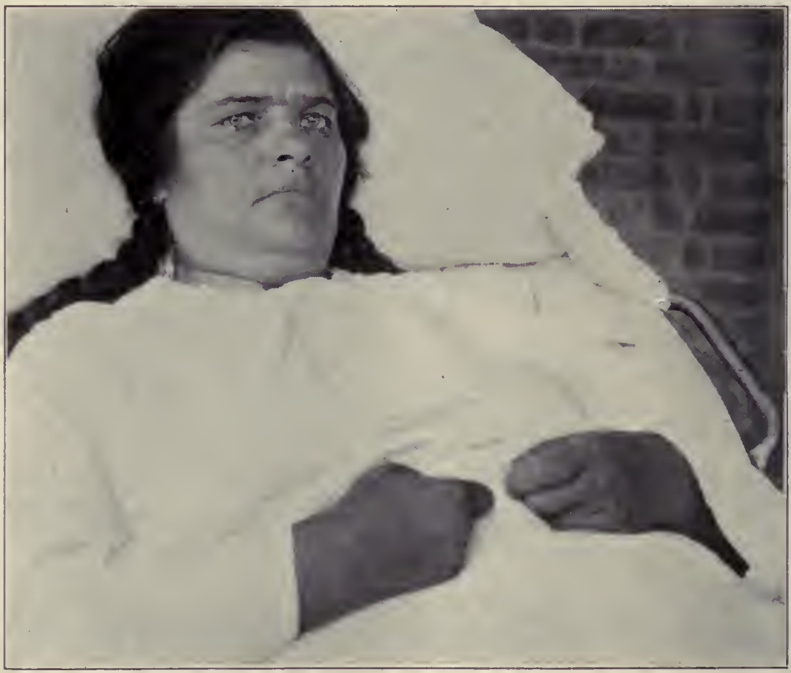

Fig. 5 (Case 15).—Choreiform type of encephalitis. A characteristic position of the hands; it resembles the driving position so frequently found in tetany.

Another extraordinary spasm, in one case, was a spasmodic, spontaneous, dorsal flexion of the right big toe with simultaneous plantar flexion of the small toes. Occasionally fanlike separation of the small toes was seen. I could find no mention in the literature of a similar phenomenon. Tilney and Howe [2] report contractions of the muscles of the big toe, but do not mention any resemblance to the Babinski phenomenon. In my patient the spasm was startlingly regular in frequency, numbering from 53 to 55 a minute. Like the other spasms in the same patient, it persisted during sleep, although to a less degree. This patient recovered. The plantar flexion of the toes ceased first, dorsal flexion of the large toe persisting for some time.

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[2] Tilney, F., and Howe, H. S.: Epidemic Encephalitis, New York, 1920, p. 16.

In a few of our patients the position of the hands was extremely interesting. In three they made the "pill rolling" movement so characteristic of paralysis agitans. Two other cases presented the attitude found in tettany (Fig. 5). The arms were held quite close to the chest, the forearms partly flexed, and the wrist flexed, while the fingers were tightly closed over the inverted thumb, simulating the driving position. The position was maintained for many days, and in one patient was present five months after discharge from the hospital. The tonicity of the contracted fingers was evident, but it could be overcome by gentle attempts. One or two of the fingers were generally involved in a continuously intermittent spasm, although the most pronounced spasm was at the wrist. The Trousseau and the Chvostek signs were absent in both cases and neither thyroid, parathyroid therapy, nor administration of calcium affected this tetany-like phenomenon.

Muscles.—Wasting was not a prominent feature but was present to a marked degree in two cases showing evidence of nerve root and cord involvement. The bones of the shoulders, particularly the scapulae, were prominent. There was marked atrophy of the muscles of the back of the neck and shoulder girdle, with occasional wavelike movements or contractions, especially in the trapezius. They showed increased galvanic irritability and anodal closing contraction equal to cathodal closing contraction. Especially in the outer part of the trapezius, the contraction was very slow to the galvanic current. The inner half of the same muscle gave normal galvanic response. There was no pain but a dull aching and sense of fatigue in the involved muscles.

I examined four patients for the myasthenic reaction. In two out of five examinations of the patient in Case 11 great weakness and atrophy of the muscles of the shoulders and back of the neck were found (Figs. 4 and 6).

SIGNS OF MENINGITIS

Sixteen of the twenty-five cases belonged to the "meningoradicular" type of the disease. Rigidity of the neck to a variable extent was present in twelve patients and in two mild opisthotonos. Dilatation of the pupils during the attempt to flex the neck was noticed in two of the patients. The Kernig sign was elicited in six patients, but it was not constantly present. A typical Babinski sign was noticed in two patients, an atypical Babinski in twelve. The Brudzinski collateral reflex, particularly his "neck sign," was observed in three patients.

MISCELLANEOUS FINDINGS

Enlargement of the spleen was always looked for but never found. Gastro-intestinal disturbance was not common, diarrhea was not observed. Gastric irritability was present in only two patients, both of whom showed evidence of vagus involvement. Obstinate constipation was present in four. Retention of urine was present in two patients—in one of four days' and in the other of six days' duration.

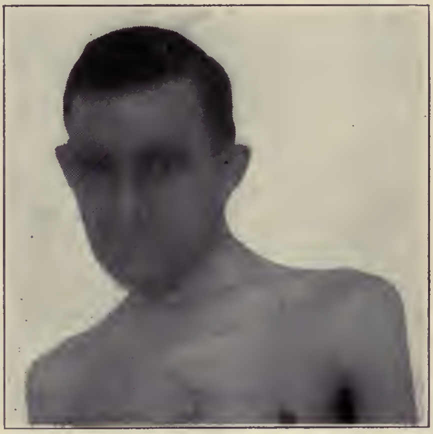


Fig. 6 (Case 11).—Paralysis of the muscles of the back of the neck. Weakness and atrophy of the muscles of the shoulder girdle are also present.

LABORATORY FINDINGS

Blood.—The red blood cells and hemoglobin showed no particular changes. If the disease was protracted, moderate secondary anemia developed. The details of the white blood counts and the differentials are tabulated in Table 1.

Chemical examination of the blood revealed nothing unusual but was an aid in differentiating the condition from other diseases in which nitrogen retention and hyperglycemia occur. As will be seen in Table 2, the blood sugar was normal in spite of the fact that in some of the cases glycosuria was present at the time.

BACTERIOLOGIC FINDINGS

The influenza bacillus of Pfeiffer was isolated from the throat in nine, and from the nose in six, out of twenty patients examined. A streptococcus was isolated from the throat twelve times and from the nose five times. Blood cultures were made in twenty cases with negative results.

The urine showed a slight trace of albumin in five cases. More than traces were not found, except in two fatal cases.

TABLE 1.—White and Differential Blood Counts in Twenty-Five Cases of Epidemic Encephalitis

Case No.	White Blood Cells	Polymorphonuclears	Lymphocytes	Mononuclears and Transitionals	Eosinophils	Basophils
1	6,400	78	14	7	1	0
2	9,700	73	19	4	4	0
3	6.200	64	25	9	2	0
4	9.100	72	18	8	1	1
5	7,400	69	22	6	3	0
6	6,300	76	14	7	2	1
7	8,100	77	14	7	2	0
8	4,100	60	30	5	5	0
9	10,300	80	15	4	1	0
10	9,600	74	17	7	1	1
11	8.800	68	23	6	3	0
12	10,100	72	23	4	1	2
13	9,000	60	30	5	5	0
14	8,700	84	10	5	1	0
15	6,600	70	18	8	4	0
16	7,400	74	19	5	1	1
17	6,600	76	16	7	1	0
18	8,100	64	26	8	2	0
19	9,100	59	36	4	0	1
20	6,800	77	11	9	2	1
21	7,400	73	13	10	4	0
22	10,200	84	12	3	1	0
23	11,800	80	11	4	5	0
24	13,800	67	23	7	3	0
25	6,500	74	15	8	3	0

TABLE 2.—Blood Chemistry Determinations in Sixteen Cases of Epidemic Encephalitis *

Case No.	Blood Urea	Sugar	Creatinin
1	14	0.12	1.12
2	12	0.07	1.0
3	18	0.09	1.2
4	10	0.11	0.9
5	12	0.12	0.8
6	10	0.07	1.2
7	9	0.09	1.0
8	8	0.08	0.9
9	11	0.12	2.0
10	10	0.13	1.1
11	12	0.07	1.3
12	14	0.09	0.8
13	10	0.07	0.91
14	8	0.08	1.2
15	16	0.06	1.2
16	13	0.10	0.92

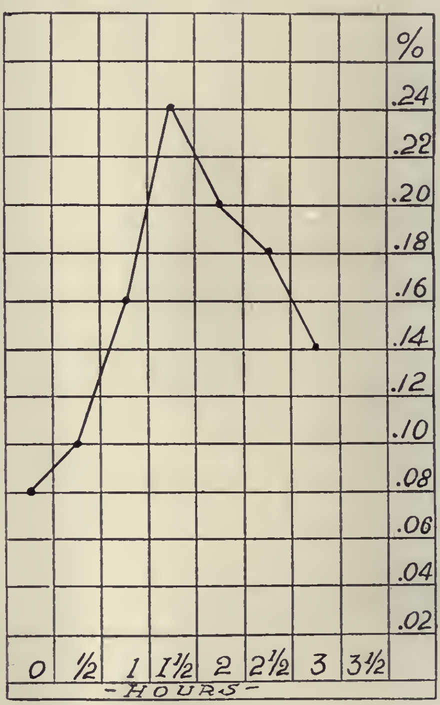
* See also Table 4.

Sugar in the Urine.—A strikingly frequent finding was sugar in the urine. It was found at some period of the illness in nine cases. The amount varied from 0.1 to 1.5 per cent. It was not present through the twenty-four hours, but no definite period, or relation to time of day or meals could be established. Sugar tolerance tests were made in four cases, and the determination of blood-sugar in sixteen cases.

There was no hyperglycemia in these cases, but all patients except those in Cases 9 and 15 had sugar in the urine at the time of the determinations. Four patients were given 100 gm. of dextrose, and blood sugar determinations were made at one-half hour intervals for three hours. In all, the sugar in the urine was increased, and there was also a considerable increase in the blood sugar which lasted for two and one-half or three hours.

TABLE 3.—Sugar Tolerance Test in a Case of Epidemic Encephalitis

Time	Sugar in Urine, Percentage	Sugar in Blood, Percentage	Remarks
8:30	0.1	0.08	Before dextrose
9.00	0.16	0.1	100 gm. dextrose given at 8:30 a. m.
9:30	0.5	0.16	
10:00	1.0	0 24	
10.30	1.0	0.2	
11:00	0.5	0.18	
11:30	1.16	0.14	



Other Elements in the Urine.—Nothing else abnormal, of any significance, was found. Acetone was present in two patients in both of whom it was associated with marked vomiting. Hyaline casts were present in the urine of the two patients whose urine had a moderate cloud of albumin.

SPINAL FLUID

The spinal fluid was clear in all cases. It was under moderately increased pressure in seventeen of the twenty-one patients on whom

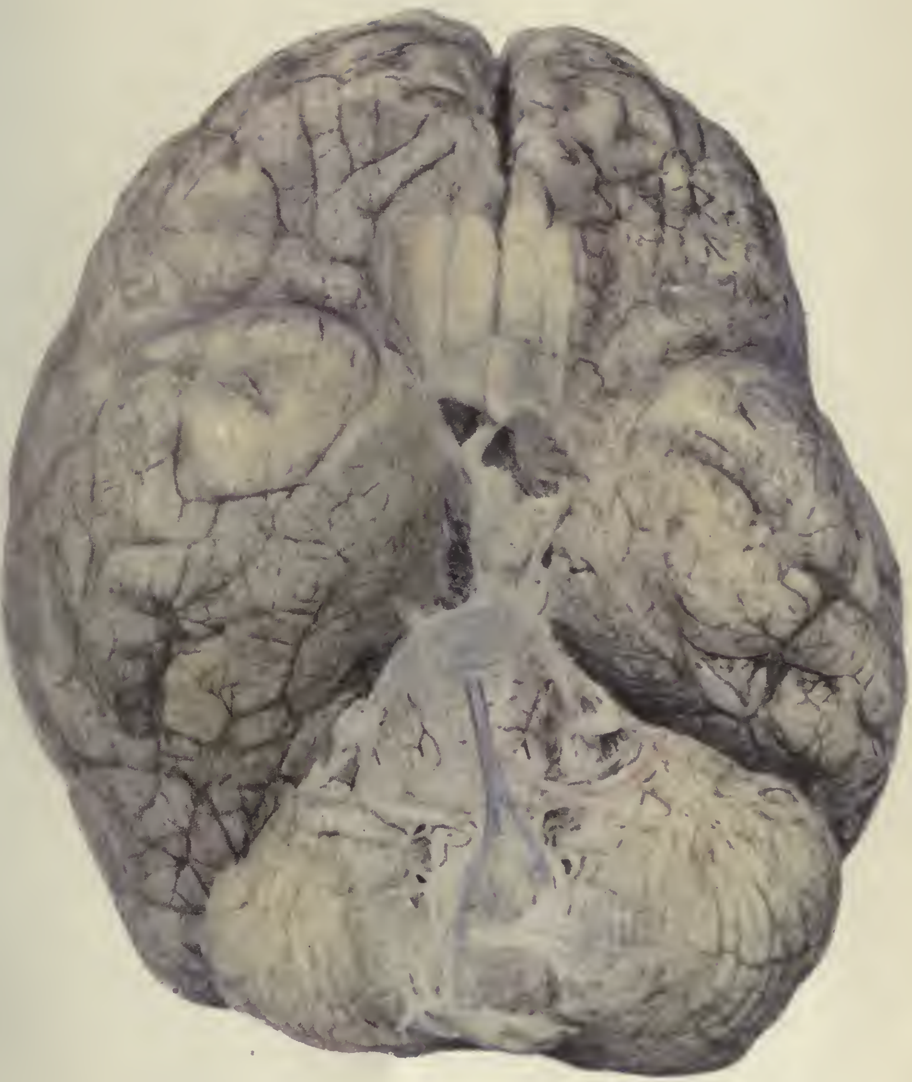


Fig. 7 (Case 20).—Base of the brain showing marked hyperemia and a moderate exudate, particularly in the region of the pons and medulla.

lumbar puncture was performed. A trace of globulin was present in all cases, and in ten there was a marked precipitate. The cell count was below 10 in two cases. No organisms were found, and the Wassermann reaction was negative in all. Further details are given in Table 4.

MISCELLANEOUS LABORATORY FINDINGS

Chemical.—Microscopic and bacteriologic examination of the stools in twelve of the cases showed nothing unusual, and the same applies to gastric analysis made in four cases. The Widal test, performed in sixteen cases, showed no agglutination.

GROSS AND MICROSCOPIC PATHOLOGY

The pathologic findings were practically confined to the central nervous system. In one case definite granular degeneration and cloudy swelling were present in various organs, but the patient had a persistently high temperature which lasted for five days. The results of postmortem bacteriologic examination of the heart blood, various viscera, brain and meninges were negative.

TABLE 4.—THE FINDINGS IN THE CEREBROSPINAL FLUID IN TWENTY-ONE CASES OF EPIDEMIC ENCEPHALITIS

Case No.	Color	Fehling's Reduction	Globulin	Cell Count	Polymorphonuclears	Lymphocytes	Bacteria	Wassermann Reaction
1	Clear	Normal	++	27	15	85	0	0
2	Clear	Normal	+++	21	7	93	0	0
3	Clear	Normal	++	30	10	90	0	0
4	Clear	Normal	+	22	13	87	0	0
5	Clear	Normal	+	14	14	86	0	0
6	Clear	Normal	++	17	10	90	0	0
7	Clear	Normal	+	9	13	87	0	0
8	Clear	Diminished	+	21	12	88	0	0
9	Clear	Normal	++	25	10	90	0	0
10	Clear	Normal	++	32	9	91	0	0
11	Clear	Normal	++	41	13	87	0	0
12	Clear	Normal	+	12	7	93	0	0
13	Clear	Diminished	++	26	2	98	0	0
14	Clear	Normal	++	17	5	95	0	0
15	Clear	Normal	+	9	10	90	0	0
16	Clear	Normal	+	10	7	93	0	0
17	Clear	Normal	+	14	13	87	0	0
18	Clear	Normal	+	16	14	86	0	0
19	Clear	Normal	++	17	6	94	0	0
20	Clear	Normal	+++	37	11	89	0	0
21	Clear	Diminished	+	22	7	93	0	0

We had occasion to examine two fetuses—both from spontaneous abortion. One was of eight months' gestation, the other of four and one half. In the older the meninges were normal, but the pia-arachnoid showed edema and congestion. There were microscopic hemorrhages in various parts of the brain and spinal cord, particularly at the base of the brain, and in the pons. Capillaries and veins were engorged and dilated, but there was no perivascular infiltration. There was also cellular degeneration, but no inflammatory changes. The whole was what one would expect in a fetus that has been dead in utero for some time. Findings in the younger fetus were similar.

In the adults the findings were the same as those described by other observers and by Allen and myself.[1] These cases, it will be recalled, were characterized clinically by marked restlessness, choreiform jerking and peculiar twitchings of the abdominal muscles, and were designated as the "meningoradicular" type. In addition to the usual findings in the brain substance, they showed pronounced changes in the meninges and spinal cord indicating that the disease was a meningo-encephalo-myelitis.

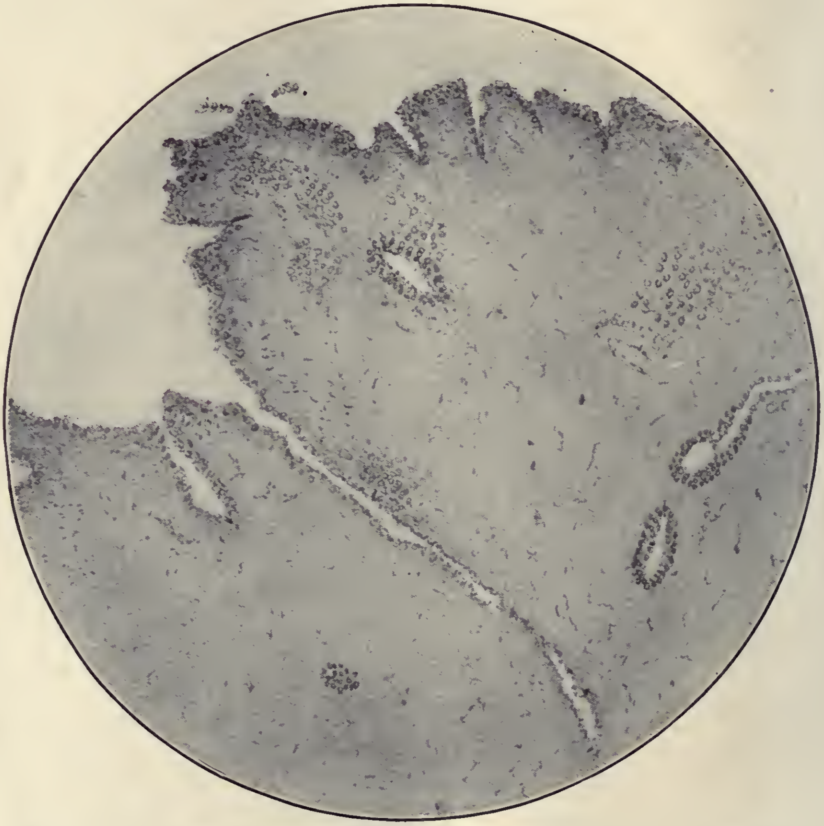


Fig. 8 (Case 20).—Base of the brain; round cell infiltration of the superficial layer; perivascular infiltration of the blood vessels.

In two of our cases the meninges showed a marked degree of inflammation. The blood vessels were engorged and dilated, and there was a considerable amount of exudate at the base of the brain (Fig. 7).

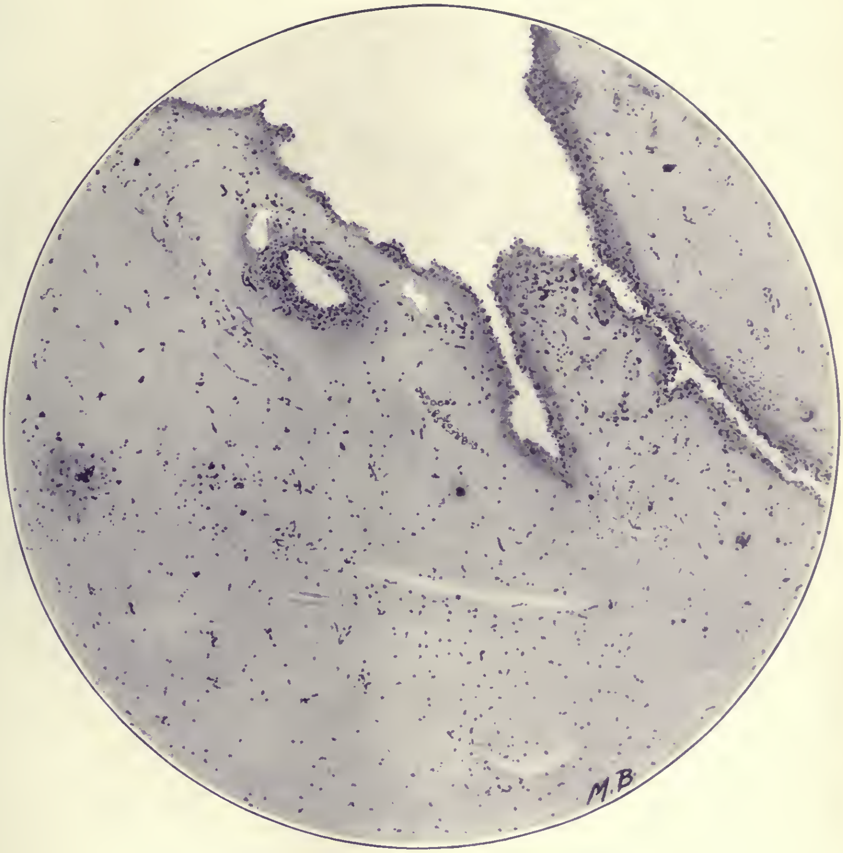


Fig. 9 (Case 24).—Base of the brain showing inflammatory involvement of the brain tissue just underneath the pia arachnoid. Note the diffuse cellular infiltration and the perivascular infiltration of the blood vessels.

The pia was thickened and grayish-red. Smears and cultures from the exudate were invariably negative. On section tiny red spots were seen scattered throughout the brain substance but were most noticeable at the base, in the pons and the cerebral peduncles, where some of the spots were quite large, being from 2 to 3 mm. in diameter. Minute hemorrhages were scattered throughout the cerebral cortex, basal ganglions, brain stem and spinal cord. The ventricles were slightly dilated and the ependyma thickened.

Microscopically the meninges showed marked inflammation, collections of inflammatory cells being found in divers parts. They were particularly frequent about the smaller blood vessels. Under high power these cells were found to be lymphocytes and mononuclears. Occasionally a polymorphonuclear leukocyte was seen in the adventitial lymph spaces.

The cortex showed slight dilatation of the capillaries and moderate cloudy swelling in some of the motor cells. The cells were swollen, the nuclei eccentric and there was some satellitosis.

At the base there was an unusual degree of inflammatory reaction just beneath the pia-arachnoid. In this region many accumulations of lymphoid cells were seen (Figs. 8 and 9). There was also diffuse infiltration with inflammatory cells. The vessels were dilated and showed marked perivascular infiltration with lymphocytes and mononuclears. The nerve cells in some places stained poorly, with some chromatolysis. In one case there was distinct inflammatory involvement of the olfactory bulb and optic nerve. The axis cylinders were swollen and stained freely and in some places were disintegrated. The protoplasm around the nuclei was increased in amount, and the nuclei were acentric. The blood vessels were congested and surrounded by mononuclear cells. In some places collections of lymphocytes and mononuclear cells were found at some distance from the blood vessels.

CLASSIFICATION

The twenty-five cases may be divided into five clinical types: (1) the lethargic type (one case), (2) the meningoradicular type (thirteen cases), (3) the paralysis agitans type (six cases), (4) the poliencephalitis type (two cases), and (5) the choreiform type (three cases).

The lethargic type was characterized by profound somnolence, cranial nerve palsies, especially of the third nerve, diplopia and masklike facies.

The meningoradicular type was marked by headache, cloudiness of sensorium, stiffness of the neck, pain along the course of the large nerves and by restlessness and twitching, often peculiar twitching of the abdominal muscles. A suggestive Kernig's sign and a pseudo or true Babinski sign were frequently seen. The spinal fluid generally had more pronounced increase of globulin and cells. Necropsy examination generally revealed more inflammation of the meninges and contiguous brain substance than in the other types.

The paralysis agitans type was characterized by a typical parkinsonian attitude and expression, various tremors and the characteristic position of the hand with "pill rolling" motion. The voice was monotonous and generally feeble. The gait was characteristic, and the patients sometimes volunteered the statement that they could run better than they could walk.

The poliencephalitis type was characterized by paralysis of one or more cranial nerves, most frequently the third and seventh. Lethargy and stupor were not prominent but marked asthenia was a feature. These cases suggested myasthenia gravis, and one patient gave a positive myasthenic reaction.

In the choreiform type the most pronounced disturbances were the extreme and persistent spasms. The patient blinked his eyes, jerked his head, twitched the mouth and made grotesque grimaces and contortions. One side of the body was generally more involved than the other. Cranial palsies were not prominent and were transient. The fever was generally quite high. Speech was thick and indistinct and deglutition difficult. The movements persisted even during sleep, although to a much less extent. Mild delirium was generally present at night but subsided toward morning.

PROGNOSIS AND CONVALESCENCE

Of the twenty-five cases in this series, four were fatal. Death apparently was due to involvement of the medullary centers. It was invariably preceded by marked disturbances in deglutition and respiration. Aggravation of the patient's condition frequently was rapid. Exceedingly few complications were seen.

Convalescence was generally slow. Exacerbations and remissions were frequent. The average duration of the disease in the mild cases was four weeks, of the moderately severe cases, about two months and of the severe cases, from three to five months. While progress is slow, it is constant. Two cases early in the epidemic were diagnosed as multiple sclerosis.

Of the twenty-one patients that survived, twelve are completely well. Five complain of lack of strength and endurance, nervousness, slight tremor and unsteadiness when excited or after moderate exertion, but they are doing some light work. The remainder are still more or less invalided—one fourteen months, and the other three, from nine to ten months after the onset of the illness. Their complaints are the same as those mentioned except to a more marked degree. Two of the patients also have cold and clammy hands and slight cyanosis of the finger tips. Three patients complain of excessive sweating and also have grooving of the nails. In two cases shedding of the nails took place.

The patients of the paralysis agitans type showed the slowest improvement. Nevertheless, of the six patients belonging to this group, all but two are well at the present time (nine to ten months after illness). Of the two, one is able to walk about and is doing a moderate amount of work. The second is still confined to bed during the greater part of the day, and after the slightest exertion exhibits generalized tremor and the characteristic position and tremor of the hands.

TREATMENT

Specific Treatment.—During the latter part of the epidemic I used convalescent serum in two cases. Twenty-five c.c. were injected intravenously, repeated in twenty-four hours. Both patients showed improvement soon after. The temperature came down to normal during the following three days and remained normal. The convalescence, however, did not seem to be shortened and the tremor, nervousness and twitchings persisted for six weeks in one case and for nine weeks in the other. The white blood count and the differential count showed no appreciable change following the injection of the serum.

General Treatment.—General care was relied on in the majority of cases. The most troublesome symptom in many cases was the dysphagia. It rendered feeding of the patient exceedingly difficult and also endangered life by the possibility of aspiration pneumonia or by suffocation. In three cases esophageal feedings were resorted to, in one case for three weeks. The patient made a complete recovery.

Sedatives.—Hypodermic injections of magnesium sulphate were tried for the twitchings and spasms but were of no benefit. Benzylbenzoate was also ineffective. Bromids and chloral were effective in large doses. Hypodermic injections of morphin and hyoscin were the most satisfactory and dram doses of paraldehyd were next in efficiency.

After the acute stage electricity and massage were of definite value.

SUMMARY

The symptoms of the patients seen in the latter part of the epidemic varied greatly from those seen early. These symptoms were restlessness, twitching and insomnia, but the transition stages and the passing of the patients in a number of these restless and twitching cases into a state of lethargy with palsies of the cranial nerves show that they are cases of lethargic encephalitis. Laboratory and necropsy findings point to the same conclusion.

While there was no relation between influenza and epidemic encephalitis in the individual cases, there was a suggestive relation between them in the community.

The symptomatology was extremely varied and resembled that of a great many neurologic diseases.

The pathologic findings consisted of minute hemorrhages in various parts of the nervous system with perivascular infiltration of the capillaries and small blood vessels with round cells and lymphocytes. In the cases belonging to the meningoradicular type these changes were apt to be prominent and considerable involvement of the meninges and superficial brain tissues was frequently present. The blood frequently showed slight eosinophilia. Sugar was frequently present in the urine. There was no hyperglycemia, but the sugar tolerance was lowered.

The mortality in this series was 16.80 per cent. The patients that survived generally had a protracted convalescence, but all showed a definite and progressive improvement.

HISTORY OF LUMBAR PUNCTURE (RACHICENTESIS)

THE OPERATION AND THE IDEA

HORACE GRAY, M.D.

BOSTON

The credit for lumbar puncture has been given in most historical reviews to one man, not always the same, generally either Corning or Quincke or Winter. The decision, however, does not seem really so simple. Many men have contributed important ideas in the growth of this subject as we know it today, and the credit given seems to have depended largely on the particular interest of each writer. I have, therefore, made an attempt to show the source of each successive new idea in this field, without in general including confirmatory work, important though that may have been.

Swifft, 1774, was stated by Wyss, 1893, to have punctured the cerebral ventricles, and the same writer noted that Wernicke and Zeuner, 1841, used the same procedure after trepanning. I have not been able to locate the original works in either the first or second series of the Index Catalogue of the Library of the Surgeon General's Office. Middeldorpf, 1856, performed a cerebral puncture on the living through a trephining, according to Levinson, 1918. Quincke, 1872, in studying the physiology of the cerebrospinal fluid in dogs, put a cannula into the subarachnoid space of the spinal cord in the upper lumbar region, and injected 1 c.c. of mercury sulphid using painters' finest granulated cinnabar rubbed up into an emulsion with Zuckerwasser and diluted with 5 to 10 volumes of water. He did not examine the fluid, however, but only the distribution of the red granules postmortem. Naunyn and Schreiber, 1881, trepanned the parietal bone and inserted a cannula into the brain, and also into the subarachnoid sac of the cauda equina, but did not make a direct puncture.

Corning, 1885, with a view to determining whether local anesthetization of the spinal cord was within the range of practical achievement, laminectomized a frog, then put a hypodermic needle through the membranes of the medulla spinalis. He concluded that this procedure was not practicable without removal of the arches of the vertebrae on account of the danger of wounding the cord, and therefore decided to inject the anesthetic (cocain hydrochlorate) between the spinous processes of the lower dorsal vertebrae. His two protocols, one on a dog and one on a man, show that anesthesia of the hind legs was in fact produced. Besides this, apparently the first direct spinal puncture, his title "Spinal

Anesthesia and Local Medication of the Cord" indicates that he was the first to perform intraspinal injection.

Rehn, 1886, demonstrated a child whom he had cured of hydrocephalus by "puncture," but he did not describe the nature of the puncture.

Wynter, 1891, somewhat as Naunyn and Schreiber did ten years before, punctured the lumbar spine and inserted a cannula on the brain. The object was continuous drainage to relieve hydrocephalus. Wynter also quoted both Hulke and P. Gould as having carried out the same procedure, after laminectomy. The author cited, Essex Wynter, appears to be that "Essex and Winter," to whom Déjerine, 1914, erroneously gave the credit for introducing puncture clinically.

USE OF LUMBAR PUNCTURE IN DIAGNOSIS

Quincke later, in 1891, first worked out a serviceable technic for clinical lumbar puncture, and since then, in the words of Nonne, 1915, "a new science has arisen." Quincke was, furthermore, the first to notice "lymph-corpuscles" in the spinal fluid. He was also the first to mention the possibility of using lumbar puncture in diagnosis, but only from the point of view of pressure and volume relations. These have remained relatively fruitless for diagnosis, though recent work is more encouraging, such as that by Sharpe, 1920, Sidbury, 1920, and Ayer (unpublished).

Lichtheim, 1893, first noted the grossly purulent and the bacteriologic nature of the fluid in meningitis following otitis media due to streptococci and pneumococci.

Von Ziemssen later, in 1893, similarly noted that "the diagnostic value of lumbar puncture is great, especially in differentiating the kinds of meningitis," and added: ". . . Just as in puncturing pleurisies." In this last analogy he antedated Widal and Ravaut, 1900, but they were instrumental in actually developing the idea by the discussion of the various kinds of cells. Von Ziemssen also seems to have been the first to report that lumbar puncture relieved headache, based on his observations on tuberculous, cerebrospinal and purulent meningitis. Von Ziemssen, like Corning, contemplated intraspinal treatment, when he said in relation to epidemic cerebrospinal meningitis: "It even seems not impossible to introduce directly curative substances in solution into the vertebral canal and so inaugurate a local therapy of the spinal cord and brain." Still a third, independent, and for a time neglected, prophet of lumbar puncture "as a means of applying medication directly to the spinal meninges," was Browning, 1894. This idea has since been put in practice by the brilliant work of many investigators.

Freyhan, 1894, seems to have been the first to describe the microscopic sediment of the cerebrospinal fluid as containing pus corpuscles and tubercle bacilli in a case which has become classic, because it again yielded tubercle bacilli in a second lumbar punctate a week later; the patient, however, made a complete recovery. A cynic has suggested that this observer misinterpreted scratches on the glass slide.

Fürbringer, 1895, in a case of tuberculosis drew off at one puncture 110 c.c., which seems a record, though the author made no such claim. Surgeons since that time have reported a liter a day draining from skull fractures.

Heubner, 1895, was the first to dare to emphasize the inestimable value of lumbar puncture "for the practitioner."

Goldscheider, 1895, independently of Corning, 1885, but in pursuance of von Ziemssen's vision of therapy, injected cocain intraspinally to relieve painful contractions due to compression myelitis, but without success.

Wentworth, 1896, in this country, found many lymphocytes in the cerebrospinal fluid of tuberculous patients, an observation often but erroneously quoted since (by Duflos, 1901; Variot, 1903; Kopetzky, 1906) as the beginning of cytology in relation to meningeal affections. The work, however, proved an important stimulus to others.

Rieken, 1895, in Quincke's clinic at Kiel was the first to perform spinal puncture in a syphilitic patient. He noted albumin but no cells, although at necropsy there was extensive softening of both hemispheres and localized pachymeningitis.

Korczyriski and Wernicki, 1896, wrote a paper on the importance of lymphocytes in serous effusions of the pleura, and related them to tuberculosis; while Bernheim and Moser, 1897, described the preponderance of mononuclears over polymorphonuclears in tuberculous meningitis, as contrasted with the reverse proportions in epidemic and purulent meningitis. These authors seem to antedate the claims of Widal and his associates, although Widal, 1901, denied the priority and the significance of the work of Korczyriski and Wernicki.

Bier and Hildebrandt, 1899, with the object of anesthetizing a larger area than could be reached by the intradermic infiltration anesthesia of Orloff, 1887, Wölfler, 1887, Link, 1888, Reclus and Wall, 1889, and Schleich, 1892 and 1894, or by the regional anesthesia of Fraenkel, 1887, Pernice and Oberst, 1890, cocainized the spinal cord through a lumbar puncture needle. This work was done without knowledge of the work of Corning, 1885, and of Goldscheider, 1895. Later Bier, 1901, though mentioning Corning denies him priority! None the less, Bier's contribution seems to be the first practical application of the idea to surgery.

Lewkowicz, 1900, showed that lymphocytosis was present in 80 per cent. of tuberculous meningitis, and seems to have been the first to suggest that syphilis (tabes or general paralysis) might also show lymphocytosis, though he did not give any evidence.

Sicard, 1900, devoted a whole thesis to intraspinal injections, discussing prior anatomic and physiologic studies on the cerebrospinal fluid, together with his own therapeutic injections of morphin, serum and other substances. He seems also to have suggested the use of intraspinal mercury, not for immediate spirocheticidal effect but in order to increase the permeability of the meninges to such drugs as were caused to circulate in the blood.

Marinesco, 1900, suggested intraspinal treatment of meningomyelitis with methylene-blue in artificial serum.

LUMBAR PUNCTURE IN SYPHILIS

Widal, Sicard and Ravaut, 1901, reported their observations, now classical, on lymphocytosis in the cerebrospinal fluid of tabetic and general paretic patients, with negative findings in paralysis agitans, chorea, hemiplegia, and other nerve conditions as controls. They drew the conclusion that lymphocytosis is "no more specific to meningo-encephalitis and tabes than to tuberculous meningitis." This view may be said to have been generally accepted, with perhaps the qualification contained in the statement of Babinski and Nageotte. Nageotte, 1901, made the important observation "there may exist in certain patients . . . a very slight syphilitic meningitis, inappreciable clinically, but well able to give lymphocytes in the cerebrospinal fluid . . . I have seen such a case." The idea was developed by Joffroy, 1901, Widal, 1902, and Ravaut, 1903. Nageotte failed at this time, however, to draw the inference that lumbar puncture should often be done despite the absence of appreciable clinical signs of meningitis. It remained for Babinski and Nageotte in 1901 to make the first statements:

> 1. Though there be an augmentation of lymphocytes in tuberculous meningitis, the diagnostic value of this sign remains complete from the viewpoint of diffuse syphilis, for it is hardly probable that doubt in diagnosis should ever arise between tuberculosis and tabes or paresis.
>
> 2. It would be interesting to puncture syphilitics systematically.
>
> 3. Besides lymphocytosis in the cerebrospinal fluid the only symptom may be an Argyll Robertson pupil.

Sicard was credited by Widal, 1901, with having stated the importance of using a fine and sharp needle in order not to tear the dura and thus allow leakage and consequent headache. Sicard reported this himself in 1902, and more recently the same subject has been discussed by McRoberts, 1918, and Wegeforth and Latham, 1919.

Milian, Crouzon, and Paris, 1902, indicated the importance of regarding headache in syphilitic patients as an indication for lumbar puncture, even in the absence of other symptoms attributable to the nervous system, because in two such cases they found an excess of lymphocytes.

Widal and Le Sourd, 1902, first reported a cure of a syphilitic lymphocytosis by the usual general antisyphilitic treatment.

Ravaut, 1903, emphasized the latent period silent clinically as the reason for his belief (which resembles that of Babinski and Nageotte, 1901) that "perhaps even before any nervous manifestation systematic cytologic examination should be made," and as evidence he stated that in a hundred and eighteen patients in the secondary stage, only seven of whom showed nerve signs, such as iritis or facial paralysis, there were excessive cells in the cerebrospinal fluid in all.

Schittenhelm, 1903, introduced the useful though not always strictly accurate terms for incipient cerebrospinal syphilis "monosymptomatic and oligosymptomatic pictures of disease or forme frustes"; and Ravaut, 1904, described lumbar puncture aptly as "veritable biopsy of the central nervous system."

Wassermann and Plaut, 1906, seem to have been the first to examine the spinal fluid of syphilitic patients by the Wassermann reaction. Similar work was carried out by Levaditi, Ravaut and Yamanouchi, 1908, who concluded that the complement-fixation method may up to a certain point be of service in the early diagnosis of nervous localizations of syphilis, and that pleocytosis is often present without the presence of lipoids which produce the Wassermann phenomenon.

Kopke, 1906 and 1907, seems to have been the first to inject arsenic intraspinally as a spirocheticide, treating sleeping sickness with atoxyl, on the basis of Castellani's discovery of *Trypanosoma gambiense* in the cerebrospinal fluid.

Ravaut, 1907, expressed the important conception that in the secondary stage syphilis behaves like a real septicemia.

Kausch, 1908, reviewed the needles, manometers and technic in general of Quincke, Von Arlt, Bier, Grosse, B. Krönig, G. Krönig, Pochhammer, Tomaschewski, Wilms, and Nemenow, and proposed a new instrumentarium.

Quincke, 1909, said that in syphilis cells occur in the fluid earlier and more frequently than in other "serous meningitides."

Alt, 1910, reported treatment of neurosyphilis by the injection of arsphenamin intramuscularly, and Iversen, 1910, intravenously, but neither examined the cerebrospinal fluid. This was then done by Sicard and Bloch, 1910, who found the Marsh ring test negative after intramuscular injection of arsphenamin, but positive after intravenous injection, wherefore they recommended the latter route.

Taege, 1910, first reported treatment of a baby by milk from the mother who had been treated with arsphenamin, and similarly Meirowsky and Hartmann, 1910, cleared up skin lesions in a child by subcutaneous injection of serum from patients treated with arsphenamin. Marinesco, 1911, was the first to report such arsphenamized serum (in vivo) intraspinally, but the first thorough appreciation of the ideal value of this preparation and the first real success with it was that reported by Swift and Ellis, 1912 (and 1913), then by Robertson, 1913.

Swift and Ellis, 1912, also tried direct intraspinal injection of arsphenamin in monkeys and thought it too dangerous to use, but injected 0.5 mg. and 1 mg. of neo-arsphenamin directly without ill effect. Wechselmann, somewhat later in 1912, used both arsphenamin and neo-arsphenamin intraspinally; Ravaut, 1913, used neo-arsphenamin intraspinally; Marinesco and Minea, 1914, also used neo-arsphenamized serum in vitro intraspinally, as did Ogilvie, 1914, and Swift, 1916.

Ravaut, 1911, emphasized that the "Wassermann reaction in the blood is often negative when gross nervous lesions are in process of evolution," and insisted on lumbar puncture without waiting for clinical symptoms.

Fraenkel, 1912, by use of the Auswertungsmethode of Hauptmann, 1910, that is, by increasing the amount of fluid from the original 0.2 c.c. to 1.0 or 1.5 c.c., found the Wassermann reaction positive in asymptomatic neurosyphilitics whose condition was similar to that of patients in whom other observers had reported negative reactions with the use of only 0.2 c.c.

Ravaut, 1913, first attempted intraspinal treatment with mercuric chlorid and cyanid. Pussen, 1913, and Marie and Levaditi, 1913, suggested the use of mercuric chlorid intracranially. Byrnes, 1914, used serum mercurialized in vitro.

Pollitzer, 1913, thought it likely that 100 per cent. of cases of syphilis would show central nervous system involvement at some early stage . . . and that discovery of this involvement depended on the methods employed. In the ensuing discussion Wile agreed.

Wile and Stokes, 1914, recommended routine lumbar puncture on syphilitic patients in primary or secondary stages. In 1915, they reported six cases of primary syphilis with abnormal cerebrospinal fluid in four.

Wile and Stokes, 1915, reported the case of a patient (Case 5) with a chancre only about one week old, yet with a positive spinal fluid.

Similarly, Read, 1915, found, only two weeks after the appearance of a chancre, which appeared twenty-three days after exposure, that the cerebrospinal fluid was markedly pathologic.

Solomon, 1916, defined "latent syphilis" as:

> A condition in which there are no discernible lesions or manifest symptoms except the positive Wassermann reaction. Syphilis may be considered a disease of remissions and exacerbations, the latent period merely a remission, following which a severe lesion is to be expected. Therefore when we treat a patient having a positive Wassermann reaction during the period of latency, we are not treating the reaction but rather the patient for the purpose of curing him of a chronic disease and thus preventing a flare-up.

He therefore affirmed that in every case showing a positive Wassermann reaction a lumbar puncture is indicated for the purpose of examining the spinal fluid. This appears to have been the earliest absolute requirement of puncture for any syphilitic condition.

Southard and Solomon, 1916, introduced the term paresis sine paresi for neurosyphilis without clinical symptoms or signs but with laboratory signs.

Thompson also, in 1916 and 1920, "urged all syphilitics, no matter at what time in the course of the disease they are seen, to submit to a spinal puncture."

Lind, in Hazen's excellent book on "Syphilis," 1919, stated that "lumbar puncture should be insisted on to the patient and his relatives whenever there is the slightest suspicion of central nervous system involvement."

Wile, 1917, reported:

> At the University Hospital (University of Michigan) specimens of spinal fluid are withdrawn from every patient having syphilis on entrance into the hospital, as a routine diagnostic measure. Many unsuspected cerebrospinal involvements have been discovered by this routine procedure.

Keyser, 1917, writing on "Syphilis of the Nervous System Without Characteristic Symptoms and Signs" made the epigram:

> The absence of characteristic symptoms or signs does not exclude syphilis . . . When in doubt suspect syphilis, and when not in doubt still suspect syphilis . . . All patients in whom syphilis of the central nervous system is suspected should be subjected to lumbar puncture.

Rosen, 1917, stated that the medical profession in general should recognize the necessity of early lumbar puncture as a routine measure in all cases of syphilis, and continued:

> At the Vanderbilt clinic it is our aim to puncture all . . . cases early, that is, after about the third month of their infection . . . Up to a year ago we were content to defer lumbar puncture, unless clinical signs of neurosyphilis were present, until just before the dismissal of the patient as cured.

Corbus, 1917, recommended that "lumbar puncture with spinal fluid examinations, be demanded in all cases of syphilis, no matter what the stage . . ."

Dennie and Smith, 1918, concluded that the cerebrospinal fluid of patients with early treated syphilis should be examined before the treatment is finished, as 50 per cent. of their patients had positive findings in the cerebrospinal fluid without signs or symptoms of involvement of the central nervous system.

Sterne, 1918, insisted that the nervous system is invaded early, and not late, as is generally supposed; and that examination of the cerebrospinal fluid along with the examination of the blood, in the early stages of the disease, is far more important than in the later stages, although such an examination is not ordinarily made.

Kaliski and Strauss, 1918, stated that: "Cerebrospinal syphilis occurs comparatively soon after infection. In patients in whom the Wassermann reaction in the blood persists, and in latent or tertiary cases in which treatment has been neglected . . . lumbar puncture should be done."

Wohlwill, 1918: "Today it must be regarded as the exception when in the stage of the general septicemia the central nervous system is not involved"; though he did not define the period of septicemia.

Frothingham, 1919: "It seems fair to demand that in all cases of old syphilis a lumbar puncture should be performed as a diagnostic procedure before instituting treatment"; and, in discussion of Frothingham's article before the Association of American Physicians, Collins made the rather reactionary reservation that "lumbar puncture will not reveal syphilis of the central nervous system as well as careful examination."

Fildes, Parnell and Maitland, 1919, recommended that the cerebrospinal fluid be examined in every case of syphilis.

Neu, 1919, recommended:

> Utilizing the serologic findings of the spinal fluid as well as of the blood in every case of syphilitic infection when first contracted and not waiting until there is other evidence of the nervous system being affected.

Eskuchen's book, Die Lumbalpunktion, 1919, gives a large amount of material, but not especially conveniently digested.

Larkin and Cornwall, 1919, demanded that study of the spinal fluid be made in every case of syphilis, as 30 per cent. of the patients show some pathologic change.

Fordyce, 1919, with his colossal experience, affirmed that more patients have serologic syphilis without objective signs than signs of syphilis without serology, hence serology is more reliable than signs.

Scott and Pearson, 1920, believe that every patient who shows a positive Wassermann reaction in the blood and every patient with late or asymptomatic syphilis should receive at least one examination of the central nervous system during the early part of his treatment. In at least eight of their 210 cases the central nervous system was involved in considerably less than a year. In fact, they feel so strongly as to say that "To be satisfied with a neurologic examination of a syphilitic as proof whether cerebrospinal syphilis exists or not, is almost criminal."

Stokes and Brehmer, 1920, remarked on the "weakness of unaided clinical judgment in the recognition of the disease," and proceeded to demonstrate in a group of thirty-nine syphilitics intensively studied that 80 per cent. had involvement of the central nervous system.

Guy, 1920: "Before any patient with syphilis is finally discharged a lumbar puncture is made."

Fordyce, 1921: "The activity of a syphilitic process in the central nervous system is indicated much earlier, more accurately and often only by the cytologic changes in the spinal fluid which should always supplement the physical examination."

SUMMARY

Generally, résumés of this field give the impression that Quincke is responsible for the technic, and that for the interpretation of cerebrospinal fluid pathology the credit is preponderantly due to Widal and Ravaut. I have attempted to trace systematically all the investigators who have initiated the successive ideas in the development of lumbar puncture,[1] and particularly to indicate the increasing insistance on early lumbar puncture.

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[1] The details of references have been omitted, since they may easily be found in the Index Medicus, the Index Catalogue, or in one or other of the authoritative textbooks, such as Hazen's.

THE PREPARATION OF COLLOIDAL GOLD SOLUTION FOR TESTING SPINAL FLUID*

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Lange's colloidal gold reaction has been found to be a highly satisfactory aid in the diagnosis of such diseases as paresis, tabes, syphilitic leptomeningitis and the like. At Bellevue Hospital, for some years past, we have been using this test as a routine measure in conjunction with the Wassermann reaction, and the result of a comparative study of the two will be published at another time. Suffice it to say for the present that, in our experience, the colloidal gold reaction is as reliable in the diagnosis of cerebrospinal syphilis as is the Wassermann, if not more so. That the test is not more widely employed is due, we believe, to technical difficulties, since even though great pains are taken, a considerable percentage of all solutions prepared are found to be useless, either because of cloudiness or excessive variations in sensitiveness. After experimenting extensively with various methods, we have found the following procedure to yield the most satisfactory results:

1. We employ 1.5 liter Florence flasks for the preparation of liter quantities. Ordinary laboratory glassware suffices; special glass is not essential. As far as cleaning the glassware is concerned, any procedure which results in a clean utensil is permissible. We prefer to treat the flasks with aqua regia and then wash them carefully several times with distilled water.

2. Triply distilled water is not necessary, provided the following procedure is followed: To 10 liters of water in a large copper still, add a few crystals of potassium permanganate and distill, rejecting the first 300 c.c.

3. In the matter of reagents, the ordinary chemically pure substances give satisfactory results.

4. The temperature may be raised either slowly with a Bunsen burner or rapidly with a rose burner, and it matters little in what sequence or at what temperature the reagents are added.

5. In our experience, contrary to that of others, we have not found it necessary to titrate the strength of the formaldehyde nor to measure carefully the amount added, but are convinced that the amount of

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* From the Chemical Laboratory, Department of Pathology, Bellevue and Allied Hospitals and of the University and Bellevue Hospital Medical College, New York.

formaldehyde used and its reaction make little difference. We use ordinary concentrated formaldehyde as purchased (40 per cent.) and add about 0.2 to 0.3 c.c. to each liter of solution.

6. An important factor, however, is the care with which the mixing is done. Rapid and thorough mixing are necessary. We advise shaking before adding the formaldehyde, and to add it while the solution is still in motion; then shake thoroughly for from one-half to one minute.

THE METHOD IN DETAIL

Into a clean 1.5 liter Florence flask place 1 liter of water, distilled as outlined in the foregoing. To this add 10 c.c. of 1 per cent. gold chlorid solution, 7 c.c. of 2 per cent. potassium carbonate and 0.5 c.c. of 1 per cent. oxalic acid. Heat this mixture to the boiling point, and at this temperature remove the flask from the flame, holding it by means of a towel and shake vigorously. While the solution is still in motion, add quickly from 0.2 to 0.3 c.c. of ordinary concentrated 40 per cent. chemically pure formaldehyde and at once shake thoroughly for from one-half to one minute. After from three to four minutes the color usually commences to develop. If, however, there should be no indication of color, the solution must again be shaken well and, while still in motion, an additional 0.1 to 0.2 c.c. of formaldehyde quickly added; this addition almost invariably produces the desired change. At no time during this process should the shaking be stopped. The color should develop rapidly to a deep red. If, however, there is a delay in the appearance of color, the mixture should be allowed to stand, and in a minute or two the color will start to develop; at this instant the solution should again be thoroughly shaken until the color reaches a deep red shade. The entire process, from the moment the solution reaches the boiling point until the color is fully developed, requires at the most about three minutes.

News and Comment

ANNUAL MEETING OF THE AMERICAN PSYCHIATRIC ASSOCIATION

At the seventy-seventh annual meeting of the American Medico-Psychological Association, held in Boston, May 31 to June 3, a new constitution was adopted, which provided—among other slight changes—a change in the name of the organization, which will hereafter be known as the American Psychiatric Association. *The American Journal of Insanity* will hereafter be the official organ of the association, and will be published under a new name—*The American Journal of Psychiatry.* The former transactions bound in book form will be omitted, but all members and Fellows will be furnished with the journal.

The Association by precedent nominates its retiring vice president for the presidency, but Dr. Sanger Brown requested that his name be withheld from the nomination, and the following officers were elected: Dr. Albert M. Barrett, Ann Arbor, Mich., president; Dr. H. W. Mitchell, Warren, Pa., vice president, and Dr. C. Floyd Haviland, Middletown, Conn., secretary-treasurer.

The time and place of next year's meeting have not been decided.

Abstracts from Current Literature

EIN NEUER CEREBRALER SYMPTOMKOMPLEX (ISOLIERTER AUSFALL DER MIMIK, PHONATION, ARTIKULATION, MASTIKATION UND DEGLUTITION BEI ERHALTENER WILLKUELICHER INNERVATION DES ORALEN MUSKELKOMPLEXES) (A NEW CEREBRAL SYNDROME [ISOLATED LOSS OF MIMIC, PHONATION, ARTICULATION, MASTICATION AND DEGLUTITION WITH RETENTION OF THE VOLUNTARY INNERVATION OF THE ORAL MUSCLE-COMPLEXES]). OSKAR FISCHER, Med. Klin. **22**:10 (Jan. 2) 1921.

Fischer reports a syndrome which he believes has not been described previously. Oppenheim in his textbook briefly reports a similar syndrome which Fischer refers to in the discussion of his case.

The patient, a man, aged 39, who about July 1, 1920, was taken with frontal headache, a week later complained of tinnitus, and about fourteen days later of a constant smell and taste of caraway. On July 29 his condition suddenly became much worse; he developed impairment in swallowing and speech with profuse salivation. During the following three days all the symptoms became markedly exaggerated, reaching the crest on August 1. There was no history of fever, but several days prior to the appearance of the paralysis the patient complained of formication on the left side of his body, also of intense yawning.

With the development of the deglutition and speech disturbance short convulsive attacks or spasms occurred. These attacks would recur about every half to one hour, so that up to forty were observed during a day.

At the height of the disturbance the patient lost the power of deglutition and speech was impossible, only a few tones resembling moans or groans being produced. Voluntary jaw movements and respiratory movements were unimpaired. Voluntary or involuntary laughing was impossible, each attempt resulting in a rudimentary facial contortion. Ocular movements were unimpaired. There was a slight nystagmus in all directions and some paresis of the left side of the face. The facial movements were slow as were movements of the tongue which, however, showed no atrophy and could be moved in all directions. There was a good pharyngeal reflex; the uvula moved normally while movements of the soft palate were noted on stimulation, but there was no coughing. Salivation was marked.

There were no other somatic findings. The reflexes, sensibility, etc., were normal. Psychically the patient was normal. The urine was normal, the blood Wassermann reaction was negative. The convulsive attacks usually lasted from fifteen to fifty seconds. An appearance of anxiety was first noted; then the head was pulled back, the mouth opened in spasm, then pulled to the right, the eyes closed, the arms flexed at the elbows and the hands became clenched. With increasing cyanosis, there appeared rather rhythmic and synchronous opening and closing of the eyes and mouth and flexion and extension of the arms, the lower limbs not being involved. During an attack no pathologic reflexes were noted, and consciousness was retained.

By August 5 the patient appeared to have practically recovered. Examination revealed a slight weakness of the left side of the jaw. Electrical reactions of facial muscles were negative.

To summarize: There was a prodromal period of several weeks with parageusia and paresthesias of the left side of the body. Later there were left sided facial paresis and difficulty in voluntary movements of the face and tongue and complete inability to speak and phonate, to chew, swallow, laugh, yawn or cough. There were profuse salivation and peculiar convulsive attacks.

The first impression was that one had to deal with an acute bulbar palsy, but the author feels that this case does not belong in either the group of bulbar or pseudobulbar palsy. Bulbar disturbance always shows involvement of motor nuclei with atrophic flaccid palsy. In the pseudobulbar cases the affection rests on involvement of the central pyramidal tracts and is only possible when there is a double-sided lesion, and then only when the cortical centers or the pyramidal fibers are injured. There is a hypertonic palsy without atrophy; there is no voluntary movement but involuntary action remains good. The facial and tongue muscles are most effected, and the pharyngeal reflex remains present; however, impairment in deglutition may occur.

It is known that the facial muscles are prone to another form of palsy in which there is retention of voluntary innervation with loss of reflex mimic action, such as laughing and crying. This is seen frequently in thalamic lesions.

Thus there are two paths for the innervation of the facial muscles: the pyramidal tract, and the extrapyramidal tracts which connect with certain thalamic centers which have to do with the coordination of mimic action. The respiratory mechanism shows a somewhat similar condition in which the automatic center is located in the medulla oblongata, while voluntary respiratory movements are cortical. Chewing, deglutition and phonation, according to Bechterew, have both a voluntary and reflex center; the reflex centers are not localized, however.

Fischer proceeds to group his findings as follows:

1. Mimic, phonation, chewing, deglutition, yawning, coughing, may be isolated and completely impaired, even with retention of voluntary function of the entire oral muscle complex. Therefore one must consider the voluntary centers and tracts of the oral musculature different from the nerve complexes which control the reflex coordination of these acts.
2. The isolated loss of these functions demonstrates the independence of the voluntary and involuntary tracts and centers.
3. The involved elements must at some place be closely connected to assume a small focus causing the trouble, as noted in this case.

Experimental work on animals has shown that the centers involved must lie between the thalamus and medulla oblongata. It would be difficult, according to the author, to place the lesion more exactly. The fact that the left facial was slighly paretic and that the parasthesias occurred on the left side of the body would indicate that the lesion was situated in the contralateral portion of the brain, but one could not state that the lesion was unilateral or bilateral.

The presence of the convulsive attacks also tends to localization. The fact that the palsy and convulsive attacks always involved the same muscles would indicate that the same focus was responsible for both troubles. Therefore a cortical lesion is not likely. The involvement of face and arms with exclusion of legs does not speak for a cortical site. The onset of the attacks with stridor, the tonic and then clonic phase would, however, point to the cortex.

Oppenheim described a case of syphilis of the pons with facial paresis, dysarthria and dysphagia, accompanied by tonic and clonic spasms of the face and neck without loss of consciousness. At necropsy Oppenheim's diagnosis could not be substantiated. According to Nothnagel, there is a spasm center in the pons. Later Ziehen and Bechterew modified this by saying that pontile lesions could only cause a tonic spasm, and that the clonic spasms were due to secondary cortical stimulation.

The author believes that the lesion in this case was in the pons, and he states that the paresthesias as noted in the case might be ascribed to the pons.

As to the etiology, Fischer believes that he has excluded syphilis, tuberculosis, hemorrhage emboli, etc. At the time an epidemic of encephalitis was present in Prague, and the author concludes that his case was one of abortive epidemic encephalitis. He realizes the lack of postmortem findings, but in conclusion states:

1. With this syndrome (once noted by Oppenheim) one has to deal with a clinical entity. 2. It has a physiologic special function for the oral musculature. 3. This apparatus may be affected as an entity.

Moersch, Rochester, Minn.

[Editor's Note.—The syndrome presented by this case is of much interest and recalls the case recorded by Dr. C. K. Mills in the *Journal of Nervous and Mental Diseases* **39**: No. 2, 1912. Unilateral mimetic paralysis with sensory symptoms, similar to those mentioned by Fischer, were present in the case of Dr. Mills. Other symptoms, however, given by the latter were wanting. In the case of Dr. Mills, the lesion was shown at necropsy to be occlusion of the anterosuperior cerebellar artery, the destruction resulting involving a portion of the cerebellum, including the superior cerebellar peduncle and also certain dorsal tracts in the pons. Fischer's case appears to be one in which these tracts were implicated, as well as some additional areas in the pons and oblongata.]

DIE ALLGEMEINE HISTOPATHOLOGISCHE CHARAKTERISIERUNG DER HEREDODEGENERATION (THE GENERAL HISTOPATHOLOGIC CHARACTERISTICS OF HEREDODEGENERATION). Karl Schaffer, Schweiz. Arch. f. Neurol. u. Psychiat. 7:193, 1920.

Heredodegenerative conditions of the nervous system are characterized by certain typical features, among which are included the familial appearance, homochronicity, homotypism, homotopism, and finally by a more or less strikingly progressive course. The classification of these disorders has always been unsatisfactory; however, the author quotes R. Bing's classification as being inclusive and yet brief. Bing divides these cases into: (1) those types in which the degenerative manifestations are predominantly motor, including the spastic and dystrophic types; (2) predominantly sensory, as in the spinal and cerebellar ataxias; (3) predominantly psychic, as in familiar idiocy; and (4) the dyskinetic types, including the myotonic, myoclonic, choreic and tremor groups.

There are innumerable transitional forms which do not permit sharp classification. According to Jendrássik, the symptom grouping is the essential feature; the origin in certain pathobiologic factors, such as heredity, familialty, consanguinity and progression, supply specific characteristics. He accordingly chose the collective appellation of heredodegeneration, considering them as being closely interrelated.

The intrinsic nature of these degenerations is quite unknown; however, the histologic findings have thus far pointed the way. The close relationship that occurs clinically would suggest a similarly close relationship along anatomic lines. Schaffer was able to exhibit the following points in support of this contention: 1. The process affects only ectodermal derivatives of the central nervous system. 2. It involves not only cell processes, but also the corresponding cell bodies. 3. It is revealed by a marked swelling, particularly of the cell body, then of the dendrites and finally of the axons.

Of the twelve cases investigated by the author, mesodermal lesions were noted in only a single instance, and in this case the lesion was circumscribed and doubtless represented an inflammatory process of exogenous origin.

Histologically, there is a swelling of the structureless hyaloplasm, resulting from the accumulation of degenerated myelinoid, protagonoid and possibly lipoid, products. In the infantile type of amaurotic idiocy, for example, the process is essentially concerned with a rapid ectodermal, that is, neuronal and neuroglial, disintegration, predetermined embryologically, since it involves the most recent phylogenetic acquisitions, and ending fatally within a year.

The important point lies in the fact that in the more chronic forms, such as the juvenile type of familial idiocy of Vogt-Spielmeyer and J. K. Walter, the process is quite similar, differing only in the extension and speed of the progress. This disease is protracted, and ends lethally only as a result of some intercurrent disease. Cerebellar heredodegenerations are essentially chronic and closely analogous. The finer changes in these include moderate swelling and simple chronic atrophy of the ganglion cells and nerve fibers, leading to a progressive cytolysis. The weblike structure of the cell body and dendrites is at first accentuated by the incrustation-like impregnations which become fragmented, first into larger particles, later into small spherical bodies, finally appearing as dust in the protoplasm. This is by no means to be identified with ordinary lipoid granules, since it is stained only with silver and therefore has been designated as "silver granular degeneration." To this process is added neuronophagia. The nucleus of the cell becomes homogeneous, deeply stained, shriveled, and ultimately melts into the cell forming a uniform mass. The picture is essentially the homologue of Nissl's chronic nerve cell degeneration. In the nerve fibers there is an irregular swelling of the myelin sheaths resulting in the formation of globules located along the course of the axons; its ability to be stained by Weigert's method is gradually lost. The axon itself shows areas of swelling, giving the appearance of a string of pearls of irregular size. This process is completed later than that affecting the myelin sheaths. The degeneration may involve only one system of neurons with their cell processes, for example, primary lateral sclerosis; or it may involve a chain of neurons, as in amyotrophic lateral sclerosis; finally, there may be such a combination as primary lateral sclerosis with degeneration of the olivocerebellar tracts. Disturbances of myelogenesis and myelonecrosis go on hand in hand. The word primary should be reserved for such degenerations as are bilaterally symmetrical, strictly parenchymatous, endogenous, and without the slightest evidence of a vascular reaction. Schaffer insists that these degenerations are invariably gangliocellular, involving first the cell, later its processes; a primary degeneration of the nerve fiber, as is assumed by Schmaus and, for instance, by Merzbacher, in aplasia axialis, has not been proved. He thinks this assumption was simply the result of the inability to detect changes in the nerve cells before modern methods came into use.

The processes noted are briefly: (1) swelling of the ganglion cells, whereby the net-like structure is thrown into relief; (2) wasting and disintegration of the Nissl granules; (3) development of hyaloplasmatic degeneration products resulting in swelling of the cell body; (4) disintegration of the perinuclear reticular structures, the so-called "silver granule degeneration"; (5) atrophy of the diseased cells, and (6) neuronophagia.

The disappearance of the Nissl granules in this process can be explained by the fact that after this material is used up the degenerated hyaloplasm cannot replenish it. Both the acute and the chronic forms have identical changes, the most characteristic of which is swelling. In an acute form, however, there is insufficient time for atrophy, which accounts for the difference in the histologic picture. The swelling itself is not specific, since it may be seen in severe infections also.

That heredodegeneration of the central nervous system is determined embryologically is proved by the fact that ectodermal structures alone are involved; also that there is a sharp selection of the structures involved, resulting in a characteristic appearance; and that phylogenetically and ontogenetically younger structures, more subject to injury, tend to be involved.

The myelodysplastic stigmas are further shown macroscopically by tectonic variabilities in the contour of the gyri of a pithecoid nature and by the occasional presence of a fetal sulcus cylindricus of His, indicating a subnormal organization; microscopically they are indicated by abnormal form and structure, such as double nuclei, abnormal localization, abnormal size and number, presence of fetal cells, abnormal slenderness of the nerve fibers, multinuclear glial formations and heterotopias.

While these diseases are both clinically and anatomically closely related, the underlying nature of them has not been determined. Such terms as deficient vitality (Jendrássik), premature senility (Bing, Higier) and wearing out of hereditarily weak tracts (Edinger) do not mean that we are any nearer the solution. The question remaining is, What influences determine the abnormal substratum? It probably can never be answered by histologic methods alone.

Woltman, Rochester, Minn.

UEBER EINE EIGENARTIGE FORM VON SCHLAFSTOERUNG IM KINDESALTER ALS SPAETSCHADEN NACH ENCEPHALITIS EPIDEMICA (A PECULIAR FORM OF SLEEP DISTURBANCE IN CHILDHOOD AS A SEQUEL TO EPIDEMIC ENCEPHALITIS).

Fritz Hofstadt, München. med. Wchnschr. **67**:1400 (Dec. 3) 1920.

During the early months of 1920 the author had occasion to study a peculiar disturbance of sleep occurring in twenty-one children varying in age from 2 years and 3 months to 13 years. In several of the cases the condition appeared following epidemic encephalitis with chorea during the patients' residence in the hospital.

The history in all cases was quite similar. For weeks or months children who previously conducted themselves normally were unable to go to sleep before 5 or 6 a. m. During the night they presented a marked motor and psychic activity, while in the daytime they conducted themselves more or less normally. Medication and other therapy were of no avail.

The conduct of the children was somewhat as follows: At the usual bedtime they would at once begin their motor restlessness, toss themselves from side to side, sit up, arrange or change the pillows, lie down again and seem prepared to go to sleep, only again to be driven to their motor activity. Some of the children would grimace, protrude the tongue, moving it about, or place their fingers in their mouth, wipe saliva over their face or spit. Some children would play with the bedclothes or tear up their own wearing apparel. One of the children, at the height of his motor activity, jumped out of bed yelling, threw himself on the floor, jumped up again, lay down, twisting about and grinding his teeth. Some of the children remained quiet; others talked to themselves, attempted to entertain themselves with their surroundings, sang, whistled, prayed or cried. The children were oriented in all spheres, gave prompt replies, but seemed to be hasty in making answers. They could give no reason for their motor restlessness, liked to sleep, were not fearful, had no pain or discomfort. When asked to remain quiet, they could do so for only a moment. The night was spent in this manner. Usually by 5 or 6 a. m., or sometimes even by 2 or 3 a. m., a deep, quiet sleep would come on, the patient sleeping until the following noon or sometimes only for three or four hours. During the day little sleep was obtained, though the surroundings were most agreeable. The children frequently went weeks, even months, with only from four to six or even fewer hours of sleep each night.

Most of the children appeared to be normal psychically during the day. Some, however, appeared to be dejected, shut-in, shy, while others were easily excited and irritable. Physically the little patients became pale, their eyes were sunken and there was loss of weight.

Hofstadt was able to observe the development of agrypnia in five cases of lethargic encephalitis while the patients were still in the clinic. He gives a brief history of each.

Nine other patients came to the clinic because of insomnia which had existed for weeks or months. The histories showed that the children had "grippe with subsequent sleeplessness," "grippe with choreiform movements," "brain grippe," all of which the author believes should be classified as epidemic encephalitis. No difference was noted in the severity or duration of the sleep disturbance as compared with the lethargic or choreic type of encephalitis. In the choreic form the interval between the appearance of the insomnia was shorter, or no interval was present.

Seven other patients had no history. Nothing was known regarding encephalitis, grip or other ailments. The insomnia and motor and psychic disturbance were said to have appeared without known cause. The symptoms and course were identical with those in the cases mentioned. The author believes that these children had, or at least harbored, the virus of encephalitis, presenting no apparent signs of the ailment at the time of onset, and that the disturbance of sleep was a secondary manifestation.

In this series of twenty-one cases the appearance of disturbance of sleep varied from being present with the encephalitis during the acute period up to seven weeks following apparent recovery. All the author's cases were noted between the months of February and the middle of May.

The agrypnia of encephalitis is so pronounced as to be characteristic. In none of the other childhood affections is such a profound change to be found, not even in the anxiety states which frequently cause marked insomnia. Severe forms of agrypnia are known to the psychiatrist, such as is found in pareses, arteriosclerosis, senile dementia and in the acute psychoses or collapse delirium. In many of the patients mental symptoms appeared during the early stages of the encephalitis, and the psychic activity of the patients during the agrypnia might be considered psychogenic, especially when the purposeless acts of the child are considered. However, the absence of serious psychic or motor manifestations during the day speaks against a psychogenic basis. Furthermore, none of the patients gave a history of a neuropsychopathic constitution.

For the present the author feels that he must pass over the question of the organic changes caused by the disease in its relation to the sleep disturbance. Whether the agrypnia depends on a disturbance of a sleep center, or is due to active toxic agents of the disease, still remains a question.

Similar disturbances of sleep as sequelae of encephalitis have been reported from Heidelberg, Meran and Vienna.

In conclusion the author states that little has been found of avail in the line of medication. The usual soporifics and sedatives are useless and even tend to increase the excitement. Small doses of veronal have at times been helpful. The best results have been obtained by means of a warm dry pack, the child being wrapped in warm dry blankets which at the same time serve as a restraint. With the production of perspiration, sleep is induced and the patient rests quietly until morning.

As to prognosis, the author has little to offer, the prognosis being much the same as that for encephalitis. The majority of the patients remain in practically the same condition after a period of six or eight weeks. In only a few cases have there been any noteworthy signs of improvement.

Moersch, Rochester, Minn.

REPAIR OF PERIPHERAL NERVE INJURIES. G. Carl Huber, Surg., Gynec. & Obst. **30**:464 (May) 1920.

The author's report covers the result of 279 operations in twenty-one series of experiments, from which 70,000 slides were made. The operating was done by Drs. Dean Lewis, J. F. Corbett, Bryan Stockey and T. Rosberg. The pyridin silver method for staining the tissues was generally used. Thirty-seven experiments were made in an attempt to prevent the formation of amputation neuromas. It was found that amputation neuromas were prevented by injecting 5 c.c. of absolute alcohol into the nerve, 0.5 to 0.8 cm. proximal to the point of section. Tangling or crossing of the neuraxes, which is ordinarily present in these neuromas, was absent in patients treated by alcohol injections. In twelve control experiments the sciatic nerve of rabbits was injected with absolute alcohol without section of the nerve. Paralysis followed but neuraxes grew down through the injected area and ultimate recovery of function resulted. From experimental results, injection of absolute alcohol did not seem to be deleterious.

The greater number of experiments dealt with nerve transplants. In seventeen cases of autotransplants of the superficial radial into a 3 cm. gap in the sciatic of dogs, using several segments of this nerve, the funicular structure of the transplant was maintained. Neuraxes penetrated the distal sciatic in experiments terminating longer than twenty-six days after the operation. Ultimate recovery of function of the muscles supplied by the sciatic occurred. Six experiments were made with homotransplants; the sciatic of one rabbit was resected and a segment of a suitable length taken from the sciatic of another rabbit and inserted into the gap. Four of the animals were under observation from seventeen to eighty-three days. In each of the experiments, downgrowing neuraxes had either penetrated or extended through the central end of the transplant and reached the distal sciatic, justifying the use of homoneuro transplant. Thirty-nine experiments deal with hetero-nerve-transplants. In the majority of these, one or two segments of the sciatic of a guinea-pig were used to bridge the defect in the sciatic of a rabbit caused by reaction. The duration of the experiments ranged from three to 358 days. Serial section showed, in cases in which neuraxes were present in the distal segment, that many of the neuraxes had passed outside the transplant and into the distal stump. The author states that while regeneration through a heteronerve-transplant may be obtained experimentally, the outcome is less certain and less satisfactory and in general requires longer time than when autonerve-transplants or homonerve transplants are employed.

In experiments with degenerated nerve transplants, it was shown that regeneration is no better than when nondegenerated transplants of the same kind are used, but that degenerated autonerve transplant and homonerve transplants may be used, if opportunity presents, with assurance of success.

In experiments with homotransplants stored in petrolatum and in liquid petrolatum at 3 C. and in 50 per cent. alcohol at room temperature, return of function in the muscles supplied by the sciatic, and downgrowth of neuraxes into the distal sciatic were obtained, showing that nerves stored in these substances may be used with success as transplants. Wrapping the suture line with various material was tried out. Cargile membrane was absorbed quickly and could be of little use. When alcoholized, these membranes were not absorbed within a period of from five to six months. Sheaths of fascia lata wrapped around the transplant caused increased proliferation of connective tissue, which the author thinks may be detrimental. In six experiments prepared carotid sheaths were used to wrap the transplant. These remained in place six months and excited less connective tissue growth than fascial sheaths. They have no advantage over alcoholized Cargile membrane. In two experiments the transplant was wrapped in a fat sheath. In the one examined 238 days after operation, the fat was replaced by dense connective tissue, thus arguing against the use of a fat sheath. In thirteen experiments an arterial tubular suture by the method of Foramitti was used to bridge defects in nerves. In eleven of these experiments kept longer than four months, downgrowing neuraxes had passed through the lumen of the artery and reached the distal nerve in which beginning regeneration was noted.

The author's summary of the results of experimental work indicates that the most favorable results are to be obtained from the use of autonerve transplants, using sensory nerves in the form of cable grafts. The type of nerve, the funicular arrangements and whether the central end is placed distally or not, are not important. Accurate end to end suture, careful technic, and a dry field are essential. Fresh homonerved transplants serve as well as autotransplants if available. Experimental work justifies the use of nerve stored in petrolatum, liquid petrolatum or 50 per cent. alcohol. Sheathing about a transplant is not necessary and serves no useful purpose although not harmful. Of the various materials used for this purpose, alcoholized Cargile membrane is the best.

Ott, Rochester, Minn.

CONSIDERATIONS SUR LA FORME HYDROCEPHALIQUE MENINGITE DE LA CEREBROSPINALE DES NOURRISSONS (THE HYDROCEPHALIC FORM OF CEREBROSPINAL MENINGITIS IN INFANTS). L. Caussade and A. Rémy, Paris méd. **11**:129 (Feb. 12) 1921.

This interesting article begins with this paragraph:

"Infection of the cerebral ventricles is a serious complication of cerebrospinal meningitis. It is a frequent cause of failure of specific serotherapy. It may occur at all ages, in adults and children. However, it is of especial interest in infants because, instead of being often clinically latent, as in adults, it has a recognizable symptomatology, and because from a physiopathologic point of view it gives information in regard to the functions of the ventricles and the neighboring cerebral nuclei. Finally, from the therapeutic point of view, the unossified state of the skull makes it possible to treat the infected ependyma by intraventricular injections of serum."

The authors review the literature briefly, recalling the various terms which have been applied to this condition: "chronic posterior basilar meningitis," "chronic cerebrospinal meningitis of the cachetic variety," "meningitic hydrocephalus," "localized cerebrospinal meningitis with ependymitis resulting in a closed cavity" and "meningitic pyocephalus." All of these terms are intended to describe a condition of cerebrospinal meningitis complicated by an acute seropurulent ependymitis, with more or less complete occlusion of the openings between the ependymal cavities and the subarachnoid spaces. The clinical syndromes resulting from this are first, ventricular hypertension, and then, hydrocephalus.

The anatomic basis of the condition is the narrowness of the openings (foramina of Monro, Magendie and Luschka) between the ventricles and the subarachnoid space in infants as compared to their size in adults. The thinnest purulent layer will suffice to block them. Clinically the condition is hard to diagnose in infants at its onset. Consequently the involvement is considerable, and walled-off foci have already formed in the arachnoid meshes before serotherapy is begun.

The authors give a case report to illustrate the symptoms of the syndrome. This emphasizes: (1) the long duration of the latent period before signs of hydrocephalus appears, (2) the masking of the condition by respiratory or gastro-intestinal symptoms or convulsions, (3) apyrexia, (4) successive stages of improvement and relapse, (5) failure to obtain abnormally large or even normal amounts of cerebrospinal fluid by lumbar puncture, (6) difficulty of injecting antimeningitic serum, (7) the presence of a lymphocytosis indicating cure in the lumbar spinal fluid combined with aggravation of the clinical picture, and (8) loss of pulsation over the large fontanel.

The authors emphasize the fact that these symptoms do not *necessarily* indicate closure of the ventricular cavities, but may be produced by obstruction of the passages from the cerebral to the spinal arachnoid spaces and that the only diagnostic signs are those directly indicative of increased tension in the ventricles. Among these are: (1) progressive increase of the volume of the skull, (2) enlargement of the fontanels, (3) spreading of the sutures, (4) softening of the skull bones (cranio-tabes), (5) pushing downward of the eyeballs, and (6) appearance of a well marked collateral venous circulation. Symptoms not mechanically due to the ventricular hypertension are: somnolence, stupor, convulsions, tetaniform rigidity and rapid emaciation.

In discussing treatment, the authors emphasized the need of intraventricular injection of serum as soon as a ventricular block is suggested by the clinical picture.

These practical suggestions are made: 1. By connecting the needle with the syringe by a piece of rubber, laceration of the brain by the needle through movement of the syringe is avoided. 2. A large amount of fluid should be withdrawn. 3. The serum to be injected should be heated to body temperature. 4. The ventricles and arachnoid spaces should be washed when possible by injecting fluid into the ventricles and allowing it to drain out through a needle inserted into the lumbar subarachnoid space. 5. Alternate the injections between the two ventricles. 6. Injections should be made only every other day. 7. Block any dripping of fluid through the hole made by the needle, by a tampon soaked in collodion.

The reader will find an article by Walter E. Dandy, entitled "The Cause of So-Called Idiopathic Hydrocephalus," published in the *Johns Hopkins Hospital Bulletin,* March, 1921, of interest in connection with this abstract.

Kraus, New York

A COMPARATIVE STUDY OF PERSONALITY TRAITS IN EARLY AND LATE DEMENTIA PRAECOX. Charles E. Rowe, State Hosp. Quart. **6**:167 (Feb.) 1921.

Of the 150 cases studied, 92 showed psychotic symptoms before the age of 30; the other group of fifty-eight cases had their mental breakdown after 30 Sixty-nine were males, of which 53 were in the group under 30 years and 16 over 30; 81 were females, 39 cases in the early group and 42 in the later. In general, the analysis was made along four lines, namely, marital condition, physical stigmas, intellectual endowment and psychic peculiarities.

As to the marital condition: Of the 92 patients under 30 years, 73 per cent. were single and 27 per cent. were married; of the 58 patients over 30, 60 per cent. were single and 40 per cent. were married.

Physical Stigmas: These include such abnormalities as weakness, small stature, youthful appearance, malformation of the cranium and of the ears, high and narrow palate, abnormal growth and distribution of hair, defective development and irregularities of the teeth. Abnormalities of this type were found in 23 per cent. of the cases, of which 18 per cent. occurred in early life and 5 per cent. in later life. The low percentage of physical abnormalities found should, the author believes, be attributed to the fact that they were not carefully recorded rather than to their absence.

Intellectual Development: The material did not lend itself readily to analysis, the patients coming as they did from locations in which educational advantages were widely varied. In making this analysis the standard followed has to a certain extent been that used by the State Department of Education, namely, that children at 14 years of age, normally bright, should be in the first year of high school, at 13 years of age in the eighth grade and at 12 years of age in the seventh grade, etc. A child two years behind this schedule is considered as making slow progress. Of the cases developing in early life 53 per cent. of the patients appear intellectually normal with 47 per cent. backward; in later life 75 per cent. were intellectually normal with 25 per cent. backward. In patients developing mental symptoms after 30, 32 per cent. of those normally intellectual were found to have had two years of high school or more and 20 per cent. were bright, but had had a limited education. Of the group in early life 20 per cent. had had two years of high school education or more and 10 per cent. normally bright, a limited educational training.

Personal Peculiarities Existing Previous to the Development of the Definite Psychic Symptoms: These are analyzed according to three types of make-up. First, the "shut-in" type which includes the shy, seclusive and the nervous, sensitive, conceited types of Kraepelin; second, the lazy, wanderer type, and third, the conscientious, docile type. In turn the shut-in type is divided into two classes, those who never have made friends and who have led a typically seclusive life and those mildly seclusive, who have had a few friends, mostly of the same sex. In this analysis of the 150 cases, I considered 48 per cent. to be definitely seclusive. This percentage is somewhat lower than that found by Hoch. Of the fifty-eight cases developing in later life, 47 per cent. were of the total cases studied were placed in the diligent, docile type, and all of cent. were included. It would appear that this definitely seclusive type prevails almost in equal proportion no matter what the age of the person at the time of the development of his mental breakdown. In the group called mildly seclusive were placed 21 per cent. of my total cases. Here this type of make-up occurred in 29 per cent. of the cases developing in later life and in only 16.5 per cent. of the cases developing in early life.

Patients considered as socially normal made up only 11 per cent. of the total number studied, or 17 cases out of 150. Of cases of mental disorder occurring in people over 30, 19 per cent. were socially active and in the cases under 30, 6.5 per cent. As to those considered of the lazy, wanderer type, 11 per cent. were found; 5 per cent. occurred in people developing dementia praecox late and 15 per cent. in patients younger than 30. Eight per cent. of the total cases studied were placed in the diligent. docile type, and all of these cases occurred in persons who showed a psychosis in early life, or 13 per cent. of the ninety-two cases at hand.

Pollock, Albany.

INTERPRETATION OF MUSCLE FUNCTION IN ITS RELATION TO INJURIES OF THE PERIPHERAL NERVES. C. C. Coleman, Surg., Gynec. & Obst. **31**:246 (Sept.) 1920.

The report is based on material collected from the neurosurgical services of General Hospital No. 11, Cape May, N. J.; and General Hospital No. 41, Staten Island, N. Y. In peripheral nerve injuries the degree of disability is determined by the loss of motor function and the presence of regeneration by return of motor function, and not by sensory loss. Motor function is easily misinterpreted, which often leads to wrong diagnoses and to the attributing of good results to operations when such are not present. In analyzing muscle function as modified or abolished by nerve lesions, the supplementary motions in the upper extremity and those possible or likely to be met with in the lower extremity, are given. In motion at the shoulder, the action of the paralyzed deltoid may be substituted rarely by the supraspinatus in conjunction with the fixation muscles of the shoulder. Flexion of the forearm in paralysis of the musculocutaneous may be performed by supplementary action of the brachioradialis. With musculocutaneous and musculospiral paralysis, the motion of the paralyzed muscles may be made possible by the action of the pronator radii tires. Extension of the forearm is seldom absent, due to the persistence of the long head of the triceps, paralysis of which is usually associated with brachial plexus injury, due to the branch to this part of the muscle being given off high up.

There is no substitution in pronation with median paralysis, but the brachioradialis brings the wrist to the neutral position but not farther. The same may be said of supination when the biceps and supinator brevis are paralyzed. Flexion of the wrist is accomplished by the action of muscles supplied by both the median .and ulnar nerve, and is possible with only one of either nerves intact. With both these nerves paralyzed, flexion may be supplemented by the extensor ossei metacarpi pollicis innervated by the musculospiral nerve. Extension of the wrist is due to the action of muscles supplied by the musculospiral. However, the hand may be elevated by strongly flexing the fingers due to tension on the slightly shortened extensor tendons. This is not possible with marked hypotonia of the extensors.

Flexion and extension of the thumbs are not effectively compensated when the primary muscles for these movements are paralyzed. Adduction of the thumb is effected chiefly by the action of abducator pollicis. Complete flexion of the fingers results from the action of muscles supplied by the median and ulnar. In combined lesions of the ulnar and median, moderate flexion of the fingers is possible by extension of the wrist. This is due to the pull on the flexor tendons caused by the position of the wrist.

In high median lesions, the patient is usually able to flex the last three fingers due to the ulnar supply to the profundus. The principal disability of median injury as regards finger movements is inability to flex the index fingers. This may be effected in some cases by extension of the wrist. Extension of the first phalanx is not possible in musculospiral paralysis, but the last two phalanges can be extended through the action of the interossei. Lateral movements of the fingers are effected chiefly by the interossei. The common extensors may spread and the long flexor approximate the fingers in supplementary action. In sciatic injuries, flexion of the leg and the thigh is usually preserved, due to the semitendinous, whose main supply is given off high up and is not injured. The quadriceps action may be substituted by the tensor fascia femoris. Inversion of the ankle, normally caused by the tibialis anticus, may be performed through supplementary action of the tibialis posticus unless the foot is held at right angles. The peroneus longus may cause plantar flexion in paralysis of the calf muscles.

OTT, Rochester, Minn.

COLONY AND PAROLE CARE FOR DEPENDENTS AND DEFECTIVES. C. BERNSTEIN, State Hosp. Quart. **6**:133 (Feb.) 1921.

Out of a total population of 2,100 inmates at the Rome State School (two thirds of which are custodial cases), over one-third, 781, are being provided for outside the institution as follows:

	Males	Females	Total
In colonies	312	237	549
On parole at home	81	46	127
On parole working	49	56	105
	442	339	781

In addition to the above, ninety-seven boys and fifty-seven girls were discharged during the year after passing through colony care and training and having therein proved themselves trustworthy were promoted as indicated in the above statistical statement to parole and finally after a successful experience on parole, for an indefinite period, depending on the reactions of the individual, to a full discharge.

The boys on farm colonies and in industrial colonies are able to make an equally satisfactory showing. The boys at the Kossuth Colony this year earned $5,120.28. They were employed variously at janitor work in stores, operating elevators, delivering goods, in coal yards, caring for lawns, furnaces, shoveling snow, assisting painters, bicycle repairing, house cleaning, etc. The boys on farm colonies under the supervision of the man and wife on the farm did the farm work and were able to produce more than enough on each farm to pay all cost of operation and in addition the boys were allowed time to assist neighboring farmers at 15 to 25 cents per hour and in this way were able to earn spending money and money for the savings bank as well as for personal clothing. This practice also serves to train and develop the boys for parole to outside farmers where they have surely been most helpful during the past summer while farm hands have been so very scarce, and thus seventy-six boys were sent out on parole in this way between April and October, 1920, and earned $1,966.47, of which $1,072.67 was expended for their current needs, and $893.80 was sent in to us and placed in the savings bank to the credit of the boys (each boy having his own individual bank account).

The boys out on parole are able to save about one-half of their earnings for the savings bank, and the girls about twenty-five per cent. (There seems to be a greater tendency to "doll up" the girls more than the boys.)

Forty girls placed out on parole during the year earned $3,138.92, of which $2,111.74 was expended by the girls for current personal expenses, clothing, etc., and $1,027.18 was saved and placed in the savings bank to the individual credit of the girls. Fourteen of these girls who had been out nearly the whole year had earned over $150 each.

There are at least two very good arguments in support of colony care for those who are subject to or in need of assistance during the period of their incapacity or enforced limited freedom and action, namely:

First: the great need for making the limited funds available reach the largest number posible, and second, rendering the enforced restriction of freedom and economic and social limitations as humane and as little humiliating as possible.

It is our experience that the unit of from sixteen to twenty-four girls or boys living in a colony under the supervision of a man and wife, or girls under a woman and an assistant, can easily earn sufficient to support the unit and in prosperous times have a little surplus for individual savings, and in this way self-respect is engendered in the individual rather than dependency, humiliation and discouragement. Rather than handing out alms we are supervising self-earned support and instructing our patients not only in hygiene and animal inhibition, but also in habits of industry and thrift and honorable self-support, the sheet anchors of moral prophylaxis.

Pollock, Albany.

DE QUELQUES TRAITMENTS DE LA MIGRAINE (CERTAIN METHODS OF TREATING MIGRAINE). P. Pagniez, Presse méd. **29**:45 (Jan. 15) 1921.

This paper discusses the comparative merits of the theories and methods of Bouché and Hustin (*Bull. de l'Acad. Royale de méd. de Belgique*, 4th Series **30**:723 [June 26] 1920), of Widal, Abrami, and Iancovesco (*Presse méd.* **28**: 893 [Dec. 11] 1920, and of Didsbury (*Progrès méd.* **35**:429 [Oct. 2] 1920).

Bouché and Hustin have found in migraine a group of complicated blood vascular symptoms (not enumerated here) that differ from the "hemoclastic crisis" (*crise hémoclasique* of Widal). These they consider to be of anaphylactic nature. They also claim that when an organism is sensitized to several substances, it no longer reacts to the injection of either of these substances, if it has just survived a shock caused by one of them; hence, if horse serum or crotalin be given in a series of injections, there will be less reaction to whatever substances are causing the migraine.

Reactions to these injections are generally local, but frequently severe, causing a rather extensive patch of erythema which may last a week. Injections are given weekly for two months, then fortnightly for from two or three months. One course may effect a permanent cure of the migraine, or a second may be required in the following year or two. If horse serum has been used in the first series, crotalin may be substituted in the second, or vice versa. One injection consists of 1 c c. horse serum or 0.1-0.2 mg. crotalin.

The mechanism of this therapy is not clearly understood. It should be reserved for cases proved refractory to other treatments. Marked disadvantages appear in the intensity of reactive phenomena that may occur, in the possible phase of aggravation, and in the chance of producing serious results hitherto unreported.

A safer and well recognized treatment consists in giving peptone (about 0.5 gm.) one hour before meals during the intervals between seizures. The resulting postponement or amelioration of attacks has often given brilliant promise at first, but end-results have been less satisfactory. Some disagreement exists as to whether the peptone merely alleviates disordered digestion, or whether it acts as a specific against this particular anaphylaxis.

Widal and his collaborators find insufficiently digested albuminoids carried in the portal vein after digestion, and stopped by the normal liver. In alimentary anaphylaxis the liver fails to function in this manner, and the phenomena of a hemoclastic crisis (sudden arterial hypotension and leukopenia) takes place. The same crisis can be produced experimentally by direct injection of these albuminoids into the vena cava. Migrainous seizures are accompanied by hemoclastic crises.

Didsbury's method is based on a conception of migraine as a chronic headache of superficial local origin. He believes that hypersensitive points and areas can always be found over the head during the intervals between attacks. These are to be massaged for thirty minutes daily. Some benefit is expected after a fortnight, and a permanent cure after continued treatments. Pagniez and others admit the existence of such cases, possibly amenable to that treatment, but contend that they are not cases of migraine.

Huddleson, New York.

ETUDE DE LA SUEUR DANS LES BLESSURES DE LA MOELLE (A STUDY OF THE SWEAT IN WOUNDS OF THE SPINAL CORD).

André-Thomas, L'Encéphale **15**:233 (April 10) 1920.

This is a most important paper bearing on the uses of the knowledge of the relations of the voluntary to the involuntary nervous systems. The author begins his article of twenty-one pages by emphasizing the frequency of the occurrence of changes in sweating, pilomotor reactions and other sympathicogenic phenomena in tramatic lesions of the spinal cord. He has already contributed articles on these subjects.[1]

He emphasizes the difficulties—the variability of the sweating in different persons and in one person at different times, as well as the influence of ordinary psychic and physical changes. André-Thomas did not care to subject severely wounded soldiers to the discomfort of a pilocarpin test, and limited his observations to cases complicated by fever or infection.

The observations were made by inspection and touch, neither litmus nor silver nitrate paper being used.

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[1] Reactions ansérines ou pilomotrices dans les lésions et les blessures du système nerveux, Paris méd., Juillet, 1918. Contribution a l'étude des centres pilomoteurs de la moelle épiniere chez l'homme, Soc. de Neurol., Dec. 5, 1918. Les réactions pilomotrices et les réflexes pilomoteurs dans les blessures de la moelle. Réflexe encéphalique et reflexe spinal. Soc. de Biol., March 29, 1919.

Two stages exist in spinal cord injuries. The first, that of shock or inhibition. In this stage sweating is absent in the paralyzed portions, or nearly absent, though the line of anesthesia and that of absence of sweating are not always the same. Sweating is only present in segments innervated by the sympathetic nerves above the lesion, that is, those which have maintained their connections with the brain. For this reason, André-Thomas speaks of sweating of brain origin (sueur encéphalique).

After the first period of shock or inhibition, a second period of overactivity of the segments below the lesion begins. With this there also occur changes in the nature of the sweating—sweating of spinal origin (sueur spinale).

The author presents a number of case reports, many with necropsy findings. He says, "The most important point to study is the level of sweating, the lower level of brain sweating, the upper level of spinal sweating, on account of the information which this sign furnishes us as to the localization of sweating centers in the sympathetic column of the spinal cord and in the diagnosis of the level of the lesion." He correlates the level of the sweat centers (involuntary nervous system) in the spinal cord segments with the level of the sensory changes (voluntary nervous system). He shows that these levels are not the same in the skin, as would readily be expected by analogy to the work of Langley on the cat. He recalls the work of Head and Riddock, published in *Brain* in 1918.

He states that the sweat centers for the head, neck and the upper part of the thorax are in segments C 8 to D 3, inclusive, and that the centers for the upper extremities lie in D 5, 6 and 7 and probably lower.

The article is worth reading in full by all who wish to better their knowledge of this method of diagnosing spinal lesions.

(The reader may refer to Gaskell's "The Involuntary Nervous System," page 38, or to the *Journal of Comparative Anatomy* **29**:424, 1918, for the anatomic basis.)

KRAUS, New York.

A PERSONALITY STUDY OF LATE DEMENTIA PRAECOX. ANNE E. PERKINS, State Hosp. Quart. **6**:174 (Feb.) 1921.

In studying the make-up and personality of thirty-eight women now in the hospital, who developed dementia praecox in the fourth and fifth decades, certain points were noticed: (1) good preservation of the personality, (2) natural affect, (3) little progression of deterioration, and (4) unnatural attitude toward marriage, with failure of adaptation to its demands. Of the entire group, thirty-three belong to the paranoid type; in these the chief characteristics may be summed up thus:

1. Twelve show remarkable preservation of the personality. They are neat in habits and appearance, industrious, more in contact with the world than the usual patient with dementia praecox and take an interest in outside affairs. Ten have never evidenced hallucinations; no mannerisms, silly laughter or masturbation have been observed, and the shallowness customary in dementia praecox is absent. Several have excellent judgment in buying, planning financially, saving money, etc., and two are not infrequently consulted about such practical matters as purchasing dress materials and designing dresses.

2. Fourteen show good emotional responsiveness, it being possible to establish with them a closer affective rapport than is commonly experienced in dementia praecox; there is less evidence of discrepancy between their ideas and mood. They are more natural, open, companionable and agreeable than patients with other types of dementia praecox.

Twelve have marked scolding reactions owing to paranoid delusions. Only three evidence depression, and at times shed tears because they wish to be at home, to earn money or to care for the family.

3. An outstanding feature is the tendency of patients with paranoid cases to continue at a certain level, with little or no progression of deterioration. Only two have undergone much deterioration. Like Kraepelin's paraphrenic types, several "do not show the deterioration in conduct or the emotional indifference so characteristic of dementia praecox. There is not the same disruption of personality because there is far less disturbance in the emotional and volitional sides of the mental life. The clinical pictures are characterized chiefly by the marked paranoid delusions. The behavior of these patients also remains well in harmony with the ideas and moods." Two might perhaps be classed as Kraepelin's confabulatory type, having delusions of royal birth, disowning relationship to parents, considering themselves queens, princesses and the bride of Christ. Two might be called the fantastic type, having a medley of extremely absurd, disconnected, bizarre delusions.

4. The unnatural attitude of the dementia praecox patient toward marriage is shown by the majority. Of the sixteen who never married, several repeatedly broke engagements, shrinking from approaching marriage; others showed marked indifference toward men. In some cases adequate history could not be obtained.

Of the twenty-two who married, nine separated from their husbands, and at least five others are known to have failed to adapt themselves successfully to marriage. Six developed the psychosis after childbirth; one left her husband shortly after the birth of her child; another definitely broke down after the birth of her second child—in the early forties. Dementia praecox often develops during the puerperal period when a woman has found adaptation difficult, for once she has borne a child, the bonds are tighter, she cannot so easily get away, hence develops a psychosis after or near childbirth. One insisted that her marriage had been annulled, always signing her maiden name and addressing her children's letters with this name. One developed a definite psychosis after an attempted sexual assault.

Pollock, Albany.

APHASIE DE WERNICKE—UNIQUE SEQUELLE D'UNE CONTUSION CEREBRALE PAR CONTRE-COUP (WERNICKE'S APHASIA—AN UNUSUAL SEQUELA TO CEREBRAL CONTUSION BY CONTRE-COUP). F. Bremer, Rev. Neurol. **27:**132 (Feb.) 1920.

The clinical picture in Bremer's case may be detailed as follows: The patient, a mason, aged 38, fell 21 feet from a scaffold, striking directly on his head and sustaining a slight scalp laceration in the midparietal region on the right, 6 cm. from the sagittal suture. When first seen by the author, twenty-four hours after the accident, the patient, although unconscious, displayed great motor restlessness associated with continuous but utterly senseless speech. Respiration was normal, the pulse rate 85, and the temperature 38.5 C. No pupillary disturbance was noted, nor could there be determined evidence indicative of basal fracture, paralysis, cerebral compression or meningitis.

No essential change was marked until the fifth day when the motor agitation was noted to have subsided considerably and sensibility to have reattained its normal status. Response to interrogation at this time, was definitely paraphasic. On the seventh day, the patient, although still bed-ridden, was observed to smile and extend his hand when approached. It was impossible, however, to secure comprehension to spoken words and replies were still paraphasic.

Lumbar puncture performed on the tenth day after the injury, showed xanthochromia and 50 cells (60 per cent. polymorphonuclears), with increased albumin but no disturbance in the chlorid content, indicating aseptic reaction to intrameningeal hemorrhage, presumably of slight degree.

Neurologic examination made on the fifteenth day, by which time the patient was entirely ambulant, revealed slight paresis of the right arm, unassociated with incoordination or adiadokokinesis. No change was noted in sensibility and, except for slight exaggeration of the wrist reflex on the right, no disturbance could be elicited in the deep or superficial reflex response. Vision was apparently normal.

Mimetic power and articulation showed no defect, but there was absolute lack of comprehension for spoken words and inability to name presented objects. Spontaneous and repetitional speech were characteristically paraphasic with evidence of periphrasis and perseveration. There was no evidence, however, of alexia or agraphia. Memory showed lacunar amnesia covering the accident and subsequent shock-period.

After a month, there was noted some improvement in the comprehension of spoken words and disappearance of the pleocytosis and the wrist jerk inequality. Roentgenologic examination of the head at this time proved completely negative and, aside from headache, there was absence of subjective somatic complaint.

Pathologically, Bremer suggests the possible dependence of this syndrome on focal hemorrhage or softening in the left cerebral cortex.

Raphael, Kalamazoo, Mich.

THE REPRODUCTIVE GLANDS AND MENTAL DISORDER, WITH SPECIAL REFERENCE TO DEMENTIA PRAECOX. George H. Kirby and Charles E. Gibbs, State Hosp. Quart. **6**:147 (Feb.) 1921.

We recently made an attempt to determine whether or not by simple physical examination of the male genitals any gross differences could be determined between dementia praecox and other psychoses. Within a few days, 313 patients were examined. The psychoses were: dementia praecox, 186; manic-depressive, 50; general paralysis, 49, and a miscellaneous group of 28. Most of the patients were taken alphabetically in several mixed wards. the diagnosis being unknown to the examiner. Only what seemed to be a definite departure from normal limits was recorded as abnormal. The testes were palpated for size and consistency.

As shown by the tabulation the dementia praecox patients showed the highest rate of variation in size, although the differences were not great. Patients with general paralysis and dementia praecox showed a definitely higher rate of changes in consistency, chiefly softening, than did patients with manic-depressive psychosis. This difference was also definite in cases in which there were changes in size and consistency in the same person.

The softening in general paralysis was more marked than in dementia praecox. These differences perhaps become more significant in the light of the age distribution, since 76 per cent. of the patients with dementia praecox were under 41 years of age, while only 28 per cent. of patients of manic-depressive psychosis and 29 per cent. with general paralysis were under 41.

The variations in size and consistency in dementia praecox were greatest at the age extremes, 100 per cent. of those under 21 and 83 per cent. of those over 41 showing some variation as to size or consistency. Although only 24

TABLE 1.—Variations in Size and Consistency of the Testes

	Number of Cases	Abnormal Size, Per Cent.	Abnormal Consistency, Per Cent.	Abnormal Size and Consistency, Per Cent.
Dementia praecox	186	56	47	31
Manic-depressive psychosis	50	46	22	12
General paralysis	49	42	58	28
	285			

Columns showing abnormal size and abnormal consistency include cases showing both changes in same person as shown in the last column.

TABLE 2.—Age Distribution

	Under 41 Per cent.
Dementia praecox	76
Manic-depressive psychosis	28
General paralysis	29

per cent. of patients with general paralysis were over 50, the impression was gained that the softening was more like that found in senile cases than was the softening in dementia praecox.

It is appreciated that this is a gross method, that some of the cases may be within normal limits, and that other factors, such as circulation and nutrition, may have a bearing.

Various other deficiencies of general development were observed in a significant number of cases, but no great differences in the three groups were noted, except that the penis was considered to be small or short in 59 per cent. of the cases of dementia praecox and in 40 per cent. of the cases of general paralysis, but in only 28 per cent. of the manic-depressive patients.

Pollock, Albany.

CLASSIFICATION OF GOITER: AN ANALYSIS OF ONE HUNDRED CASES. Carrington Williams, Am. J. Med. Sc. **161**:223 (Feb.) 1921.

The author's material was the patients admitted to the surgical clinic of St. Luke's Hospital of Richmond, Va. He divides the cases into three groups. Group 1 contains the cases of simple goiter. In this group there were sixty-one patients of whom fifty-seven were females and four males. The duration of the goiter varied from two months to twenty-five years with an average of seven and one-half years. This group of patients had practically no symptoms except those of pressure.

Group 2 presented an interesting picture. The patients were middle-aged and had had goiter for a long time, with no symptoms. The goiter suddenly began to grow, and the patients became restless and nervous and had symptoms of hyperthyroidism, but did not have a tremor or exophthalmos. There were twenty patients in this group, of whom nineteen were females. The age incidence was 34 to 58 years with an average of 47 years. The duration was from two months to thirty years with an average of eleven years. Seventy-seven per cent. of the cases gave a history of recent growth. The pathologic condition in this group was that of the simple goiter class.

Group 3 embraced the patients with the classic symptoms of exophthalmic goiter. There were nineteen patients in this group, all females. The age incidence was from 15 to 51 years with an average of 33 years. The duration of symptoms was from two months to seven years, with an average of two years. Fifty-seven per cent. of these cases gave a history of a recent growth of the goiter. The pathology in this group was hyperplastic parenchymatous goiter which is composed of closely packed tubules lined with multiple rows of large cells with little or no colloid material. Williams believes that surgery is the cure for all the groups. He says that roentgen-ray and radium treatment should be limited to group 3, and that it is conceivable that in the future the radiologist may take preference over the surgeon in the treatment of group 3.

Wilson, Philadelphia.

LE MECANISME DU HOQUET (THE MECHANISM OF HICCUP).

H. Roger and E. Schulmann, Presse méd. **29**:161 (Feb. 26) 1921.

Graphic records were made of one of the "epidemic hiccup" cases, discovering several interesting features not ordinarily observed clinically. The subject was in the third day of the disease; typical hiccup occurred at every third or fourth breath, and caused vomiting on ingestion of any substance. His general condition was good in spite of fatigue, hunger and insomia. A remission of the hiccup took place for several hours during the investigation.

The apparatus was set to show inspiration on the ascending stroke. The curves (illustrated in the article) showed that each characteristic spasmodic inspiration was immediately preceded by a quick forced expiration, unnoticed clinically. The expiratory spasm almost always began at the end of inspiration, but sometimes during the expiratory pause or the beginning of inspiration.

Abortive spasms were not uncommon; occasionally the inspiratory spasm was lacking, exceptionally the expiratory. Both abortive and complete spasms were reduplicated at times.

During the period of hiccuping, respirations were irregular and unequal, with an amplitude from one-third to two-thirds of normal, and a rate about one and one-half times normal. During the period of remission, some abnormalities still persisted, for example, inspiration was longer, and the expiratory pause shorter, than normal, and one abortive hiccup was registered.

Fluoroscopy in the myoclonic phase (period of hiccuping) showed a transitory tetanizing of the diaphragmatic arches. Instead of contracting normally they were agitated by fibrillary twitchings. No attempt was made in this paper to theorize on the paths of the nervous impulses involved.

Huddleson, New York.

HEMIHYPERTROPHIE CONGENITALE DES MEMBRES ET DU PAVILLON DE L'OREILLE CORRESPONDANT (CONGENITAL HEMIHYPERTROPHY OF THE EXTREMITIES AND IPSOLATERAL PINNA).

C. Roubier, Rev. Neurol. **27**:147 (Feb.) 1920.

Roubier directs attention to the ubiquity of the minor or so-called physiologic asymmetries of limb size and contour in contradistinction to the distinctly infrequent occurrence of true congenital hemihypertrophy. The latter may be of three types: (1) complete, involving an entire half of the body or at least the arm and leg, (2) partial, involving a single limb, and (3) facial, involving one side of the face. Of these three types, the first, of which the author's case is an example, is perhaps the most uncommon, not more than forty such cases having been reported to date in the extensive literature available on this subject.

The case presented by Roubier was that of a single, young adult soldier with apparently negative family and personal history except for bodily asymmetry dating from birth. Examination revealed the presence of definite hemihypertrophy (including both skeletal and soft tissues) which involved both extremities on the right and the corresponding pinna, but apparently did not affect the facial elements or the trunk. There was no integumentary abnormality on either side or evidence of adiposis. The superficial veins, however, were quite prominent, particularly on the right, and there was additionally noted, on this side, slight cyanosis of both extremities with edema extending over the leg and ankle. The temperature also, over this leg, was somewhat higher than over the left. No neurologic disturbance could be detected except the apparent diminution of muscular force on the right. Examination of the special sense organs and viscera was completely negative, and it was equally impossible to elicit evidence suggestive of psychic disorder.

Roubier considers the various hypotheses which have been advanced as to the etiology of this condition, commenting briefly on the possibility of congenital hemilateral vasomotor imbalance or paresis, of venous obstruction dependent on fetal malposition, and of fundamental metameric disturbance or faulty mesodermic development during embryonal life, but concludes, finally, that none of these conjectures are validly tenable and that the actual causative mechanism must still be regarded as preeminently obscure.

The article is illustrated with three excellent figures, and there is appended an extremely comprehensive bibliography.

Raphael, Kalamazoo, Mich.

EPILESSIA, ANAFILASSI E DISTIROIDISMO (EPILEPSY, ANAPHYLAXIS, AND DYSTHYROIDISM. V. M. Buscamo, Schweiz. Arch. f. Neurol. & Psychiat. **7**:2-304, 1921.

The author calls attention to his publication, in 1915, in which he first formulated the theory that idiopathic epilepsy is nothing more than an anaphylactic crisis provoked by the presence in the blood of certain abnormal proteins of thyroid origin. He also assumed that the so-called symptomatic epilepsies, e.g., those of arteriosclerosis, general paralysis of the insane, had the same origin, basing his theory at that time on a certain number of cases studied clinically and anatomo-pathologically in which he always found an abnormal protein crystalline in form in the thyroid colloid, coincidently with lymphocytosis, etc. Since his first article no proof has been brought against his theory, but many authors, of whom he cites twenty or more, have reported facts corroborating his theory, some experimental and others clinical.

In proof of his theory he now presents a total of 100 cases in which the thyroid was examined with care. Of these patients, thirty-nine were epileptics or persons having epileptiform seizures and the other sixty-one were not epileptic or suffering from epileptiform seizures. He offers this interesting result: The protein crystals were present in the thyroids of 84 per cent. of the epileptics or those suffering from epileptiform seizures and in 40 per cent. of the thyroids of nonepileptics and persons not suffering from epileptiform seizures. He suggests as treatment for epilepsy partial thyroidectomy, with successive administrations of thyroid extract, in order to eliminate the production of the abnormal protein. He mentions Crile's removal of one suprarenal and three-fourths of the thyroid (also section of the cervical sympathetic) as analogous to his suggestion for treatment although arrived at from another point of view. A full bibliography is appended.

Gurd, Ann Arbor, Mich.

A SKETCH OF THE ORIGIN OF THE CEREBRAL HEMISPHERES. C. Judson Herrick, J. Comp. Neurol. **32**:429 (Feb. 15) 1921.

The endbrain (telencephalon) of lower vertebrates is dominated by the olfactory system, and the differentiation of this region in all higher forms appears to have taken place largely under the influence of various systems of nonolfactory fibers which have grown forward into this olfactory territory. Increasingly complex correlations of the various other senses with smell have involved the elaboration of separate correlation centers in the forebrain for each of these reflex patterns, different in each species of animal according to its mode of life. The diverse patterns of cerebral architecture exhibited in the vertebrate series are, therefore, structural expressions of these functional relationships, namely, the various parts played by the olfactory and the different nonolfactory systems. Illustrations are given of the way in which this principle works out in the lower vertebrates in shaping the form of the cerebral hemisphere, and the attempt is made to reconstruct the probable history of these form changes in early Devonian times when the amphibia with fully formed cerebral hemispheres arose from ganoid fishes with more primitive brain structure, and the functional factors which operated in effecting these morphologic changes.

Herrick, Chicago.

RECHERCHES SUR LA VALEUR DE L'ELECTRODIAGNOSTIC DANS LE DIAGNOSTIC DE LA TETANIE DE L'ADULTE (RESEARCH REGARDING THE VALUE OF ELECTRODIAGNOSIS IN TETANY OF THE ADULT). F. Naville, Ann. de méd. **8**:280 (Oct.) 1920.

The author carried out electrical reactions on three groups of cases, twelve of tetany of assured diagnosis, twelve doubtful cases and twenty of conditions clearly different in nature. He found the ulnar nerve most advantageous for these tests and recommends its continued use for the sake of standardization. The findings reaffirm the state of electrical hyperexcitability in tetany.

In an adult with tetany, either manifest or latent, a muscle twitch response is elicited by the negative closing current (negative pole on the ulnar nerve) with less than 1 milliampere; by the positive closing with less than 2 milliamperes; by the negative opening and by the positive opening by less than 5 milliamperes. A state of tetanization of the muscles supplied by the nerve is reached in the tetany case by a negative closing current under 5 milliamperes and by a positive closing current under 10 milliamperes.

In the cases without tetany, the negative closing reading was almost always above 1 milliampere, the positive closing above 2 milliamperes, the negative closing tetanization threshold always above 5 milliamperes.

The negative closing tetanization response with less than 5 milliamperes and a negative opening muscle twitch under 5 milliamperes are conclusive proof of tetany. The other alterations are suggestive but not pathognomonic of the disease.

Davis, New York.

THE CURABILITY OF TUBERCULOUS MENINGITIS. Francis Harbitz, Am. J. Med. Sc. **161**:212 (Feb.) 1921.

The author calls attention to the curability of tuberculosis in other parts of the body, namely: the lungs, bones, glands, kidneys and serous membranes. There is one tuberculous condition that is still considered hopeless, namely, tuberculous meningitis and tuberculosis in the central nervous system. Harbitz thinks we should review our ideas on the subject, and says that as long ago as 1858 Rilliet wrote about the healing of tuberculous meningitis. In 1914, Bokay collected twenty-nine cases, in all of which the tubercle bacillus was demonstrated in the spinal fluid or by the inoculation of guinea-pigs. To date there have been about forty cases of tuberculous meningitis with recovery. Harbitz reports a case in which at postmortem there were found evidences of a previous tuberculous meningitis and encephalitis with numerous tubercles on the surface of the brain as well as in the interior of the ventricles; section of the spinal cord showed areas of lymphocytic infiltration in the leptomeninges with here and there, particularly in the lumbar cord, distinct tubercles which were in the process of healing.

Wilson, Philadelphia.

ETUDE HISTO-PATHOLOGIQUE DES CENTRES NERVEUX DANS TROIS CAS DE MALADIE DE FRIEDREICH (HISTOPATHOLOGIC STUDY OF THE NEURAXES IN THREE CASES OF FRIEDREICH'S ATAXIA). G. Marinesco and C. Tretiakoff, Rev. Neurol. 27:113 (Feb.) 1920.

Marinesco and Tretiakoff, in their cases, found pathologic changes in both the white and gray matter of the cord. The changes in the white matter, particularly marked in the dorsal region, were essentially atrophic in nature, showing involvement of the posterior columns (including both endogenous and exogenous fibers) and the pyramidal tracts, as well as the bundles of Flechsig and Gowers. As regards alteration in the gray substance, there was noted, characteristically, evidence of slowly progressive simple atrophy in the cell bodies and fibers of the columns of Clarke, the posterior horns, and the posterior root ganglions. In the latter, there was additionally remarked traces of regenerative effort in the form of pericellular and, somewhat less frequently, periaxional plexi. The authors feel the gray matter disturbance to be basically dependent on primary changes occurring in the white substance, i. e., the process is essentially cellulipetal rather than cellulifugal.

No trace was detected in the cord of actual cellular disintegration or infiltrative reaction although a certain degree of apparent inflammatory reaction was determined in the cerebral and cerebellar cortices, which the authors regard as more or less incidental and wholly secondary to the process in the cord.

Comment is made on the characteristic reduction in gross volume noted in the cord, medulla and cerebellum which seems to stand in direct relation to the age of onset and the clinical duration of the disease, thus indicating, in the authors' conception, its basically developmental nature and its probable dependence on alteration (biochemical rather than inflammatory) in the myelin and neuraxial material of the centripetal neurons and ascending fiber tracts.

Raphael, Kalamazoo, Mich.

SUR LA PRESSION DU LIQUIDE CEPHALO-RACHIDIEN (SPINAL FLUID PRESSURE). J. A. Barré and R. Schrapf, Bull méd. 35:63 (Jan. 19 and 22) 1921.

The writers conclude that the maximal normal pressure of the spinal fluid, when measured with the Claude manometer, is 20 cm. (of water) for the lying position and 40 cm. for the seated position with head lowered. They consider as pathologic any pressure exceeding 25 cm. in the first position and 45 cm. in the second. The lifting of the head in the seated position raised the pressure from 5 to 10 points.

To prevent agitation and its resultant effects on pulse and respiration the writers follow the custom of giving a hypodermic injection of sedol ten minutes before the puncture and using ethyl chlorid as a local anesthetic.

They believe that occasionally in spite of the needle being properly within the subarachnoid space, the fluid does not flow due to certain sufficiently vague circulatory alterations induced by fright and other emotions. Reassurances or the inhalation of a stimulant, such as ether, in such instances initiates the flow.

The absence of a spinal fluid "pulse" synchronous with the arterial pulsations is probably indicative of spinal fluid hypertension.

Davis, New York.

PARALYSIE DU NERF SPINAL PAR FRACTURE DE LA BASE DU CRANE (PARALYSIS OF THE SPINAL ACCESSORY NERVE FOLLOWING BASAL SKULL FRACTURE). G. L. Regard, Rev. Neurol. **27**:141 (Feb.) 1920.

Regard's patient was an artilleryman, aged 38, who had fallen from a loft. When first seen, several hours after the accident, the patient was deeply comatose and examination, at this time, revealed complete areflexia, with the exception of greatly diminished knee jerks, profound depression in the sensorimotor field, myosis, marked facial pallor, frigidity of the extremities and bradycardia (60). Although it was impossible to determine primary evidence of cranial injury or depression, there was noted copious bleeding from both ears (particularly the left) and from the nose. Prosopoplegia was present on the left and lumbar puncture yielded blood-stained fluid. A small abrasion was noted on one thumb.

By the eighth day sufficient improvement had been made to permit a more exact neurologic examination. The original reflex and sensorimotor disturbance was now no longer in evidence but there was definite persistence of the left facial paralysis (subgeniculate type) with complete loss of function in the left ear. It was apparent, also, that the spinal accessory nerve on this side had suffered total paralysis, as shown by the raucous, nasalized voice, paralysis of the left palate and left sterno-cleido-mastoid and trapezius palsies.

Tetanus developed on the thirteenth day and terminated fatally six days later, the infection entry having presumably taken place through the abrasion on the thumb. Necropsy was not performed.

On the basis of the picture presented clinically, the author feels justified in the assumption of basal skull fracture, specifically of the petrous portion of the left temporal bone of the posterior longitudinal type.

Raphael, Kalamazoo, Mich.

LE TRAITEMENT DE L'ENCEPHALITE LETHARGIQUE (TREATMENT OF LETHARGIC ENCEPHALITIS). L. Cheinisse, Presse méd. **29**:146 (Feb. 19) 1921.

"Therapeutic nihilism" is discarded in favor of a method originated by Pic and approved by Netter (Traitement de l'encéphalite lethargique, *Bulletin de l'Academie de Médecine,* March 30, 1920). Most other methods that gained some repute are disapproved. There is evidence that the serum of convalescent cases is harmful rather than beneficial. Autoserotherapy has not proved valuable.

In the recommended method, 1 to 2 c.c. (adult dose) of oil of turpentine, preferably old, is injected into the cellular tissue of the abdominal wall. An abscess should form, and is to be opened, when fluctuation is established. If no abscess develops after the first injection, a second is given two days later, and a third after another two days' interval if required. Urotropin by mouth or intravenously is also recommended as a supplementary medication but not as a substitute. The abscess treatment should be started as soon as the diagnosis is certain. Urotropin may be given later.

Huddleson, New York.

GERODERMIE CHEZ UN ENFANT (GERODERMIA IN A CHILD). P. Haushalter, Rev. Neurol. **27**:15 (Jan.) 1920.

Report is made of a case (a boy, aged 3) in which the onset was traced to a severe attack of pneumonia at the age of 1. Otherwise, the personal and family history were negative, except for the occurrence of goiter in the mother.

Clinically, the gross picture was typical of gerodermia. In addition to the characteristic senile expression and integumentary change, however, there was noted prominence of the parietal bosces, "Gothic" palate, elongated pinnae, orchitic hypoplasia and low-pitched voice. There was no indication of dwarfism, the body length being 92.7 cm. Mentally, except for a certain gravity of manner and seriousness of interests, there was apparently no determinable evidence of deviation.

Haushalter calls attention to the fact that in most of the cases described in the literature, the condition seems to have occurred in older persons, and to have been additionally characterized by dwarfism, alopecia and often by pronounced asthenia. The probability of the existence of causative endocrine imbalance in these cases is indicated, also the desirability of their inclusion under the collective clinical designaton, genito-glandular dystrophy, as recently suggested by Souza and Castro.

Raphael, Kalamazoo, Mich.

CORTICAL WORD BLINDNESS. Hubert S. Howe, Neurol. Bull. **3**:64 (Feb.) 1921.

The author reports the case of a man aged 55. After a peculiar attack of a feeling of being smothered, which lasted several hours, he suddenly found that he could not read his newspaper. He was not unconscious, and there were no other complaints. General physical and neurologic examinations were practically negative. His vision was 20/40 in both eyes, and the fields were normal, showing no hemianopsia. His speech and intelligence were not impaired. He could read single letters fairly well; he could copy; but he could not read whole words or sentences or write spontaneously. The author considers this case as showing pure cortical word blindness, and places the lesion in the angular gyrus. The case was exceptionally well studied clinically, and apparently represents a rare instance of a dissociated defect due to a circumscribed lesion, and as such is worthy of record.

Wechsler, New York.

L'ESCARRE ET L'ATROPHIE CEREBRALE DES AFFAIBLIS INTELLECTUELS (TROPHIC SORES AND CEREBRAL ATROPHY IN THOSE WITH MENTAL DISORDERS). Henri Danaye, L'Encéphale **15**:681 (Dec. 10) 1920.

This author calls attention to the coincidence of trophic sores (bedsores) with changes in the brain in cases of psychosis which are plainly of toxi-infectious origin and associated with demonstrable pathologic changes in the brain itself. On the other hand, patients with psychosis unassociated with demonstrable pathologic changes do not suffer from trophic sores. Of these, examples are dementia praecox vera and epilepsy. Of the conditions showing organic change, those which most often show complicating trophic sores are general paresis and senile dementia. In both of these profound anatomic changes of the brain are present.

Kraus, New York.

THE NERVOUS SYSTEM OF A TWO-HEADED PIG EMBRYO. Mabel Bishop, J. Comp. Neurol. **32**:379 (Feb. 15) 1921.

This is a detailed anatomic description of the brain and nerves of a laterally symmetrical dicephalous pig embryo of 22 mm. The lateral sides of the brain and peripheral nerves are normal; the conjoined medial sides show various degrees of duplicity from completely separate and normal relations of the snouts to complete union into a single individual in the trunk region. A study is made of the regulatory phenomena in the conjoined regions, and all of the anatomic evidence points toward a merely regulatory adjustment in a healthy, orderly developed, though teratologic, embryo, for wherever normality or near-normality can assert itself, it is anatomically realized or approximated. The paper is illustrated by twenty illustrations of wax reconstructions and microscopic sections.

Herrick, Chicago.

CRANIAL DEVELOPMENT FOLLOWING ENUCLEATION IN EARLY YOUTH—POSSIBLE EFFECT IN ADULT LIFE. Gelford Dickinson, Am. J. Ophth. **4**:270 (April) 1921.

In the case cited, in early enucleation of the eye, the boney structure of the orbit continues to grow—especially in those parts that afford the most protection normally, the superior and temporal portions. This abnormal growth is out of proportion to that of the other parts of the orbit. The superior growth is most marked showing an increased frontal sinus to about double its size.

The possible effects in later life are focal infection and neuralgia of the ophthalmic branch of the fifth nerve.

DeLong, Philadelphia.

Society Transactions

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Regular Monthly Meeting, March 17, 1921

James B. Ayer, M.D., *President, in the Chair*

The Neurological Department of the Boston City Hospital presented these papers:

REPORT OF A CASE OF CERVICAL SYRINGOMYELIA. Dr. J. J. Thomas.

A Pole, 46 years of age, who could speak little English, gave a vague history of paralysis of one side eight years before, from which he recovered. Since that time he had had headache and dizziness, which had become more constant the past seven months, with occasional vomiting, and some trouble with vision. On entrance into the Boston City Hospital he complained of headache and pains in the neck, arms and shoulders, dizziness and staggering as if drunk, and constant tinnitus in the left ear. Examination revealed slight tremor and considerable ataxia of both arms and legs, more marked on the left, and unsteadiness in standing, with a tendency to fall backward and to the left. There was also constant marked nystagmus, which was slow when the eyes were turned to the left, and rapid when looking to the right, and the man complained of diplopia on looking to the right and to the left with one eye as well as when both were open, which was probably due to confusion of vision. The pupils and fundi were normal. The reflexes in the arms were lively and equal. The abdominal reflexes were slightly diminished on the left, and the knee and ankle reflexes were increased and greater on the left. At times ankle clonus was obtained on both sides, and at others only on the left. For a short time Babinski's sign was present on the left, this and the ankle clonus disappearing again. There was apparently hypalgesia and hypesthesia on the lower right side of the face, and the man insisted that he felt nothing on the body below the clavicles. Both labyrinths were normal for the caloric tests, and hearing was normal with tests by tuning forks, except that there was some diminution for both high and low tones on the left, and bone conduction was slightly lowered for low tones on the right. The tympanic membrane and conduction apparatus were apparently normal in both ears. There was slowness of rotation of both forearms. The Wassermann test on both blood and spinal fluid was negative. The spinal fluid was under slightly increased pressure, globulin was increased, later normal, the cells varied in number on different occasions from 5 to 10 per c.mm. The colloidal gold test was negative. Roentgen-ray examination of the head and cervical spine was negative.

An operation was advised, and a subtentorial decompression was done. There was well marked increase of the pressure, but no growth was found in the cerebellum or on the auditory nerve. At the close of the operation the pulse rose rapidly, probably from the effect on the vagus nerve, and the patient died soon after removal from the operating table.

Necropsy revealed nothing remarkable in the internal organs. In the brain the frontal lobes were slightly adherent to each other, and the occipital lobes somewhat flattened, the left more. Section showed no growth in the brain or cerebellum. The upper part of the cord was thin and flattened, and on section showed a cavity centrally located. This cavity at its greatest diameter, which was in the upper cervical region and transverse in direction, was 8 mm., gradually diminishing upward to end in the medulla, and extended below into the upper dorsal region, the length of the cavity being 18 cm. Microscopically the cavity showed the usual aspects of syringomyelia. The cavity in the lower cervical regions was centrally located, and extended back dorsad in the median line nearly to the dorsal surface of the cord, with little lateral involvement. Higher in the cervical region the cavity extended laterally with some involvement of the column of Clark. Still higher there was splitting of the cavity into two, and at other points as many as five, cavities, some always central, but at places extending into the dorsal horns of the cord. The cavities showed in places ependymal cells at the margin. The symptoms, though uncommon in syringomyelia, were probably to be explained by involvement of the restiform body in the medulla, or the column of Clark.

DISCUSSION

Dr. C. M. Campbell thought that the possibility of some associated condition with the syringomyelia ought to be kept in mind, and mentioned the case of a woman in her fortieth year who had been epileptic since 16. She had been taking large doses of bromid and was alcoholic. Since adolescence she had been having frequent convulsions. The sensory tests showed no anomalies of sensation. The patient did not complain of headache, and there was no choked disk. She became increasingly duller. Finally choked disk appeared, and the patient died. At necropsy, in addition to definite syringomyelia, a glioma was found in the posterior part of the brain.

Dr. H. I. Gosline mentioned a case which showed in serial sections the splitting of the cavity as it went upward, and he thought that if all these patients had lived long enough the same peculiarity might have been found. Some observers of syringomyelia have believed that in certain cases other causes than glioma have played a part. In Dr. Thomas' case there was at least a borderline cell count and globulin increase and perhaps some transitory symptoms that might be due to edema. The theory of infection in these cases is not as well supported as the tumor theory. It would be interesting to know whether there was any cellular infiltration in this case of Dr. Thomas.

Dr. J. J. Thomas, in closing, stated that the roentgen ray had not been tried, and that he should not expect it to accomplish much except in cases having diffuse gliosis with formation of cavities. The case reported showed no infiltration about the vessels. From the sections one would probably classify it as a case of abnormal closure of the central canal. The cavity running back from the central canal toward the dorsal septum of the cord with dense neuroglia about it and proliferation of new blood vessels in the tissue shows that it was distinctly a congenital failure of closure. In regard to the existence of two cavities higher up, it is not uncommon to see a great many branches in such a condition, and the distance they run is variable, particularly in the upper medulla. The sections did not show any massing of gliomatous tissue at any one place, simply a thickened layer about the cavities. They showed nothing like a true glioma or even diffuse gliosis.

TYPES OF EPIDEMIC ENCEPHALITIS. Dr. A. W. Fairbanks.

No arbitrary classification can be made. The cases vary widely in the different types. The original conception of the affection is entirely changed in the light of a survey of subsequent clinical experience. Cases seen up to the present time may be grouped as follows:

1. The simple type, characterized by somnolence and lethargy, usually commencing with subjective disturbances of vision, such as blurring of vision and diplopia, but not characterized by definite objective paralysis of the cranial nerves.

2. The common type, in which somnolence and lethargy are associated with definite objective cranial nerve paralysis (our original conception of the disease).

3. The type that commences with general malaise and headache, in which somnolence and lethargy are either absent or appear as late phenomena, but in which cranial nerve palsies may be conspicuous. This type is attended by a feeling of marked muscular weakness, usually forcing the victim to bed, but no paralysis is found on examination. Weakness probably is due to interference with conduction through the motor tracts (pyramids) or more frequently perhaps to interference with conduction through the tracts for muscle-sense in the interolivary region of the medulla. In these cases well marked ataxia in the limbs is often to be found.

4. The bulbospinal type (including those sometimes termed the radicular type). Neuralgic pain is the dominant symptom and usually ushers in the disease. Its character, although usually limited, is strongly suggestive of root irritation. For instance, a case may begin with complaint of intense pain in one hand, later extending up the arm, across the shoulders and down the other arm, thence perhaps down the body; or it may remain unilateral. These patients are likely to develop delirium, often within a few hours of the appearance of the pain, or perhaps some days later. Cervical rigidity is often present, and not infrequently considerable spasticity in the limbs. Somnolence may be absent, or it may appear late in the course of the affection; and the same is true of cranial nerve involvement. Delirium, restlessness and pain dominate the picture.

5. The spastic type without pain. Asthenia is often a conspicuous feature of these cases, and stupor is usually very evident in the early period. Onset may be sudden. Somnolence may be marked for many weeks but often only in the earlier period. Flexibilitas cerea may occur in these cases. Cranial nerve palsy may occur, but as a rule is not prolonged or conspicuous.

6. Myoclonic type. Rhythmical or irregular muscular twitching, more or less general, including the trunk and abdominal muscles. An interesting and singular form in this type is the respiratory form, in which the respiratory disturbance dominates all other symptoms. In these patients the respiration may show a rhythm synchronous with the twitchings, as in an instance cited by the reader, in which for ten weeks extremely rapid respiration persisted.

7. Paralysis agitans type. Onset usually occurs with vague feeling of malaise and general weakness, and often an indescribable sense of discomfort in the legs, soon followed by coarse tremor of the paralysis agitans type.

8. Abortive type. Malaise, headache and often fleeting pains in various parts of the body may appear suddenly and be accompanied by blurring of vision and diplopia. Cranial nerve palsy may appear within a few hours and disappear almost as suddenly as it appeared. Light somnolence is usually present but brief. The duration may be brief but much weakness may remain.

DISCUSSION

Dr. J. W. Courtney stated that the more cases he saw, the stronger his conviction grew that encephalitis is merely the most striking and dangerous symptom of a general constitutional infection. This is well shown by the recent pathologic work of Dr. Harris of the Worcester State Hospital. All patients examined by her showed uniform changes in the spleen and other organs. Dr. Courtney believed that the life history of the filtrable bacillus which causes so-called encephalitis is analogous to that of *Malaria plasmodium.* He has in a given case seen evidence of recurrent activity of the organism at intervals of several months.

Dr. Harold E. Foster, who has been quantitating the sugar content of spinal fluid, reported particularly on a series of cases in which a definite diagnosis of encephalitis was made. In all of the cases except one a definite increase from the normal was noted. In some this increase was nearly twice the normal average and in one fatal case was, eleven hours antemortem, over double the usual reading, being equivalent to a high normal blood sugar. Another case showed a marked rise during an exacerbation of the clinical picture. The blood sugar was normal in all of the correlations made. The ages ranged from youth to old age. Determinations were made from the second day of the disease up to about six weeks. The blood and spinal fluid Wassermann reactions were all negative. There was nothing constant in the cell count, total protein content or gold solution curve. A study of the series would indicate that the increase in spinal fluid sugar is distinctive and in direct relation to the severity of the clinical symptoms but has no relation to the age of the patient or the duration of the disease.

Dr. A. W. Fairbanks, in closing, said that necropsies had been performed on myoclonic cases but no distinctive changes had been found. Almost all spinal fluids showed some increase in lymphocytes, from 5 to 106. The globulin had been practically always increased during the active stage of the disease. The pressure was sometimes increased, but more often not. Approximately seventy-five or eighty cases had been seen. One case in particular showed very well the bulbar syndrome. The asthenia was marked from the start; there was no paralysis but there was marked ataxia in all four extremities. There was no trace of pyramidal trouble. Dr. Fairbanks differed with Dr. Courtney as to the constitutional theory of the disease. He thought the nerve feature the dominant one.

ANHEDONIA; A SYNDROME. Dr. A. Myerson.

Years ago Ribot described the case of a young woman suffering from hepatic disease in which the main mental symptom was the disappearance of satisfaction or happiness. This symptom he called anhedonia. William James in his book, "The Varieties of Religious Experience," declares it to be a passive melancholy in that while there is no dejection or depression, there is loss of zest for living.

For two years a study has been made of psychiatric and neurologic patients from the suggestion thus given by Ribot and James, and the conception of a syndrome has been formed which may be called anhedonia, but which is more inclusive than that of the distinguished Frenchman and American.

Anhedonia is not a disease; it is a syndrome and is made up of four principal changes: First, there is a disappearance of or interference with the great organic sensations—loss of appetite, in many cases loss of thirst. There is almost invariably insomnia, and generally inability to rest well, so that the patient becomes restless. Usually the patient complains of feeling tired, but when closely questioned this fatigue is not the ordinary normal fatigue; it is a painful sense of incapacity, lethargy or "nervelessness." This last arises, so it seems, from a dropping out of that fundamental organic sensation, the impulse to activity which is one of the primitive organic kinesthetic sensations.

Second, there is loss of hunger, thirst, etc. There is loss of desire for activity in any form, although the patient is restless. This restlessness never takes the form of organized activity. Sometimes, because there is desire for pleasure and the sensations hitherto so pleasant, the individual becomes a pleasure seeker without definite desire except to fill in the painful gap in his mental life. The loss of desire and the lack of the feeling of energy cause ambition to disappear or to be impaired. This dropping out of ambition is exceedingly painful to the patient and deepens the feeling that something has happened to him.

Third, there is loss of the feeling of satisfaction which is the central symptom of anhedonia, as described by Ribot. The patients I have in mind are not definitely depressed; they are not self-accusatory; they do not weep or wring their hands or moan. They complain bitterly that nothing gives them pleasure. Food has lost its savor, drink gives no pleasure, sexual relationship gives but the veriest shadow of the former satisfaction; nothing seems worth while, though it is realized that it is not the fault of things but the fault of the individual's capacity for satisfaction. This is not the dissatisfaction of persons whose desires are not gratified. In such persons desires, strivings and wishes are keen, but in the anhedonic patient there is neither desire nor satisfaction.

Fourth, a curious accompaniment of the anhedonic syndrome which, of course, is not at all limited to it but quite common in mental disease, is the increased diffusion of excitement. Long ago Lotze pointed out that the sick or deficient organism reacts as a whole to excitement much as a child does, whereas the adult, strongly developed organism limits excitement, makes it local. Beard lays great stress on this phenomenon, without so far as I can see, knowing that Lotze had preceded him. Such persons start at a noise; they become confused when many impressions are received at once since every impression produces excitement. They are confused and excited when they meet people in groups, etc., and this excitement is painful because they become self-conscious and develop a feeling of inferiority. In some cases the patient becomes seclusive, not because he is unsocial by nature or because he hates, but because the excitement engendered by meeting people is painful, and his reaction is to avoid pain and the feeling of inferiority. The emotions are readily stirred in a disagreeable direction, and to many of these patients a sense of unreality develops, which is very painful. Doubt and obsession arise out of the feeling of unreality.

The anhedonic syndrome is common, is met after severe illness, often gradually developing long after the illness has passed. It is met in persons in whom some central ambition or desire has been so completely balked as to make achievement or the growth of personal dignity impossible. It is common in the returned soldier. He complains bitterly of loss of appetite, sleeplessness, loss of energy, loss of pleasure, loss of desire and increased excitability under the circumstances named. The most conclusive case seen was in an early stage of dementia praecox. It is often seen as a cyclic disease. Without ordinary depression, without retardation, there is simply a periodic loss of ambition, desire, satisfaction and the loss of the great organic sensations of sleep, appetite, sex desire, etc.

The etiology, it is believed, lies in changes of some sort, produced in the springs of desire and satisfaction; to wit, in the vegetative nervous system, the endocrine glands and the viscera. The study of desire and the attendant phenomena here described is believed to be fully as important to psychiatry as the study of intelligence and emotions. For whether one subscribes to hedonism or not, it is certain that the human being seeks satisfaction for his desires, and that desire is largely based on instinctive mechanisms of a prime order which constitute the foundation of human personality.

DISCUSSION

Dr. J. W. Courtney remarked that an observation like Dr. Myerson's is a signal advance in the comprehension of morbid mental states. The sensory and sensorial disorders which underlie the anhedonic state are not measurable by the esthesiometer or any other such crude instrument. They nevertheless denote an adynamic condition so marked in intensity that the perception and diffusion of stimuli necessary for apperception cease. When the anhedonism reaches a certain degree of profundity the feeling of reality is lost, and it is only by constant argumentation that the victim of this feeling succeeds in convincing himself that it is *his* hand with which he performs certain movements, *his* feet which carry him about, *his* voice with which he speaks. In a still more advanced state of depression the patient fails to convince himself by argument, and harbors the delusion that the various parts of his body are not real living parts, that he is nonexistent; and he finally carries his notions of nihilism to the point where they embrace all the phenomena of the universe.

OBSERVATIONS ON THE TREATMENT OF PARESIS. Dr. LeRoy A. Luce.

Arsphenamin and arsphenamized serum have been used at the Boston City Hospital in the treatment of about 500 patients with neurosyphilis during the past nine years. Two thousand intradural serum injections were given by either the Swift-Ellis or the Ogilvie method; these being the favored methods. No severe reactions or deaths followed. Dr. Luce presented two cases of paresis that had received a considerable amount of treatment extending over a long period of time.

Case A: A man, 42 years of age, gave a history of chancre fifteen years before symptoms of nerve involvement began to appear. Hallucinations of sight and hearing and mental deterioration predominated. The blood and spinal fluid were positive, globulin positive, cell count 46, and the colloidal gold test showed a paretic curve. This patient received seventy Swift-Ellis serum treatments extending over eight years. The blood and spinal fluid remained Wassermann-fast throughout the whole period of treatment, and the colloidal gold test did not change from the paretic curve. The cell count changed from 46 to 3.

Case B: A man, 35 years of age, with a history of chancre five years before symptoms of nerve involvement, had not received proper and efficient antisyphilitic treatment during the primary and secondary stages of the disease. He had delusions of the expansive type and was mentally exalted and erotic. The blood and spinal fluid were positive, and the colloidal gold test showed a paretic curve. This patient had received seventy-five treatments in seven years. The blood and spinal fluid remained Wassermann-fast, globulin positive, and the colloidal gold test did not change from the paretic curve.

These patients with undoubted paresis received about the same number of treatments. One man was able to do simple work, while the other was helpless both physically and mentally. These observations were reported to bring up a discussion on the value of treatment in paresis.

DISCUSSION

Dr. A. W. Fairbanks expressed the opinion that from a clinical point of view to treat a definite case of paresis was a waste of good material and more valuable time.

Dr. A. H. Ruggles mentioned three patients who had left the hospital in the past year, all of whom were capable of working, one now with negative findings and two with positive findings. On the other hand, he had observed two patients, neither of whom had had any treatment, one of seventeen years' duration and one of twenty-one years, both of whom are still alive.

Dr. F. H. Packard stated that the results in treating general paralytics at the McLean Hospital had not been encouraging; indeed, treated patients had done worse and gone down hill faster than most of the untreated ones. None of the treated patients had had remissions while a considerable percentage of the untreated patients had not only held up for considerable periods, but some of them had had strikingly good remissions of considerable duration—remissions so good as to make optimistic relatives feel that they had recovered.

Dr. H. C. Solomon stated that an analysis of the figures of state hospitals in Massachusetts show that up to 1916 about 2.5 per cent. of the patients with general paresis who were untreated had remissions, none of which lasted one year, and of the treated patients 25 per cent. had remissions some lasting for four, five or seven years. These figures indicate the value of treatment of patients whose cases had been diagnosed as general paresis.

REPORT OF A FEW CASES OF CRANIAL NERVE PARALYSIS OCCURRING DURING AN EPIDEMIC OF ACUTE POLIOMYELITIS. Drs. Robert H. Courtney and William E. Savage.

Of twenty-four patients with acute poliomyelitis who were admitted to the Boston City Hospital during the four months ending Dec. 1, 1920, five showed involvement of the bulbar region alone, one of the bulbar and a segment of the cervical region, and eighteen had purely spinal types of the disease.

Of the five patients with bulbar cases four were males and one a female. The ages in four cases were between 11 and 13 years, and in one was 3½ years. Three of the patients recovered but with permanent (five months after onset) cranial nerve paralysis; two died, one from involvement of the respiratory center of bronchopneumonia.

Two of the patients showed complete paralysis of the lower branches of one facial nerve and weakness of the upper branches. The other three showed involvement of the glossopharyngeal nerve with regurgitation of food through the nose. In these, laryngeal diphtheria was excluded both clinically and bacteriologically. The spinal fluid of all showed an increased cell count varying from 80 to 200 with a slight preponderance of lymphocytes. The treatment of all except one consisted of lumbar puncture repeated at intervals of two days. In one case convalescent serum was given, but improvement had already begun.

Necropsy was obtained in one case. Gross examination of the brain and spinal cord was negative. Microscopically there were extensive perivascular lymphoid cell infiltration and fairly diffuse and focal infiltration of endothelial leukocytes about the small capillaries of the pons and medulla. The pathologist reported a microscopic diagnosis of anterior poliomyelitis.

NEW YORK NEUROLOGICAL SOCIETY

Three Hundred and Eighty-Eighth Regular Meeting, April 5, 1921

Foster Kennedy, M.D., *President, in the Chair*

A CASE OF POLYCYTHEMIA WITH NERVOUS SYMPTOMS. Dr. Moses Keschner.

Dr. Keschner presented a patient suffering from this disease. Polycythemia, or erythremia, is a condition of the blood in which the number of red blood corpuscles is increased above 5,000,000 per cubic millimeter. It may occur as a result of physiologic stimuli or in disease as a result of pathologic stimuli. It may be relative, that is, without real overproduction of red cells. In absolute or true polycythemia the total number of red cells in the body is actually increased. This is a disease of unknown origin characterized by persistent erythremia due, it is thought, to excessive erythroblastic activity of the bone marrow, or to a failure of destruction of the red blood corpuscles. Some also ascribe it to faulty internal secretions, especially of the suprarenals. The nervous manifestations have hitherto received little attention, in contrast to those of the anemias. The symptoms are: headache, dizziness, visual disturbances, paralysis (motor and sensory), pain, muscular twitchings, convulsions, speech disorders, mental, vasomotor and trophic disturbances. There is general or localized pain, a vague aching and feeling of fulness and throbbing over the entire body, especially in the head. Some of the patients show defects of memory and general symptoms of neurasthenia; they are emotionally unstable and have a tendency to become easily depressed or excited and at times are delirious. Insomnia is a common symptom. In the vasomotor sphere, hyperhidrosis is frequently found.

Eye symptoms are common and frequently the first noted. Some patients complain of easily induced fatigue from the use of the eyes. Palsies of the external ocular muscles are quite common. These may give rise to diplopia. Scintillating scotomas, transient blindness and hemianopsia are not infrequent. Ascher has described in this disease a form of asthenopia without refractive errors, in which the patients give a history of repeated but unsuccessful efforts to get proper adjustments of glasses. The ophthalmoscope reveals changes in the fundi which are characteristic of the disease. The veins are distended and engorged so that they appear much larger and darker than the arteries; they are tortuous and may show varicosities. The disk may be normal, or slightly reddened, or it may have the appearance of mild optic neuritis.

The nervous symptoms in the anemias are due to degeneration of the neurons brought about by toxemia. The pathologic bases of the nervous symptoms in polycythemia are vascular. They may be thrombotic or hemorrhagic, and cause softening which is not due to arteriosclerosis.

The patient, a 30 year old Russian tailor, was admitted to the Mount Sinai Hospital complaining of headache and salivation from which he had been suffering for the last eight years. The salivation, as well as the headache, was almost constant. His symptoms had been attacks of tonsillitis in the past, headache, salivation, dizziness, vertigo, blurred vision, anorexia, hemorrhoids, congestion of face and eyes, insomnia, uncontrollable hemorrhage after tonsillectomy, flushes of heat and cold, tinnitus and epileptiform seizures. Blood examination revealed: hemoglobin, 170 per cent. (Kuttner); 9,800,000 red blood corpuscles; 340,000 blood platelets, and 8,000 white blood corpuscles. The red cells were microcytes and showed some slight central pallor. A later examination revealed: hemoglobin, 170 per cent. (Kuttner); 11,456,000 red blood cells; 410,000 blood platelets, and 7,600 white blood corpuscles. The color of the blood stream in the veins of both disks was reported to be dark purple, much darker than normal. The arteries were normal. The visual fields were normal. The patient was treated with atropin until there was marked dryness of the throat and dilatation of the pupil, but the salivation did not diminish, nor did local astringents applied to the mouth have any effect on it. After the withdrawal of 10 ounces of blood from the median basilic vein, the patient stated that his headache and dizziness were slightly relieved.

DISCUSSION

Dr. B. Sachs asked concerning the patient's subjective state. Dr. Keschner resummarized the chief symptoms. The patient, when asked if he felt dizzy or sleepy, replied in the affirmative. Dr. Sachs asked how the disease could be recognized.

Dr. Keschner said that it was recognized in this patient by salivation, headache, attacks of dizziness and epileptiform seizures. It was first suspected when great difficulty was experienced in drawing blood from the vein in trying the Wassermann test. There was no evidence of other nervous disturbances. The appearance of the veins in the disks and the headache were the main factors. Dr. Keschner's theory was that the salivation was central. It was not affected by atropin. The saliva had not been examined.

Dr. C. L. Dana said that a diagnosis of polycythemia could not be made from any nervous symptom.

Dr. J. H. Leiner said that in the last six months he had seen two cases of this disease in which there were no nervous manifestations at all. An increase in blood cells was present. The throat was curiously stippled like the throat in scarlet fever. There were present a black pigmentation along the mucosa, a characteristic congested eye and a cyanotic flush of the face. A woman, 55 years of age, who had polycythemia vera, had high blood pressure. Some of the skin conditions he thought were endocrine in origin.

Dr. Rosenthal, by invitation, said that a different type of polycythemia was present with the high blood pressure. It was described a long time ago by Geissler and was called polycythemia hypertonica. Here the blood pressure and pigmentation were not so marked. In these cases tuberculosis must always be ruled out, since polycythemia might be secondary to it.

Dr. Keschner said that the disease had been treated by roentgen-ray exposure of the spleen and of all of the long bones, and had been accompanied by improvement. Some writers also report encouraging results from this treatment; others say that some of these patients get better without treatment, and that the improvement simply represents a spontaneous remission. The injection of epinephrin causes an increase in the red blood cells. The condition is by some thought to be due to an adrenalemia.

COMBINED NEUROLOGIC PERCUSSION HAMMER. Dr. J. H. Leiner.

Dr. Leiner demonstrated a percussion hammer half the size of the ordinary hammer when closed, a little shorter when open. The design is that of the Roman battle axe; the balance is delicately adjusted, and it is weighted by metal and a strip of soft cushion rubber to prevent trauma. The back had originally been made of a soft piece of rubber. It is now replaced by a hard piece set in by a set screw. The original hammer also turned from side to side and swerved. In this model, therefore, a groove has been cut in the bottom of the shank with a pin running through it. At the other end of the hammer there are three tubes to contain the instruments commonly used. These are a needle (very sharp) which can be pushed out by a little button, a colored orange stick to test color vision, which snaps back when not in use, and a camel's hair brush for touch. This hammer obviates the necessity of always carrying pins with one. It can be used for making the Babinski test, also as a tongue depressor. It makes absorbent cotton unnecessary. These hammers are made by N. A. Low and Co., New York, and cost $7.50 each.

DISCUSSION

Dr. Sachs said that he had been using this instrument for the past weeks and found it convenient.

A CONTRIBUTION TO THE HISTOPATHOLOGY OF PORENCEPHALUS. Dr. J. H. Globus.

This paper will appear in a future issue.

DISCUSSION

Dr. B. Sachs felt that this work corroborated a suspicion which he had expressed about thirty years before. It still remains a question whether all cases can be explained by such defects. The occlusion of one of the branches of the larger arteries is not unknown. He asked whether Dr. Globus had sufficient evidence to show that the porencephalic defect occurred during the prenatal period or whether it could have been developed after birth.

Dr. Foster Kennedy said that only a few days ago he had seen a child with a definite nuclear aplasia of the right seventh and hypoplasia of the right sixth nerves. The mother had been desperately ill with influenza at six months' pregnancy. At the time he had thought that the failure to develop was due to encephalitis in the fetus.

Dr. Globus, in answer to Dr. Sach's question, again stated that there was absolutely no history of illness in the postnatal life of the child. The mother had had uterine inflammatory condition early in pregnancy and before conception. Lesions both chronic and subacute had been found which therefore must have taken a long period to develop. The disease must be placed in the later part of intra-uterine life or in early childhood.

SOME FEATURES OF THE NEURO-PSYCHIATRIC WORK ON ELLIS ISLAND. Dr. B. Onuf.

Dr. Onuf said that this was the first time that the question of immigration had been brought up before the Society. The aim of Ellis Island was to control admissions to the United States, to exclude undesirables and to restrict the entry of those who might become public charges. The question of undesirability, from the medical side, involved personal shortcomings and disabilities, transferable diseases and hereditary factors. Laws provide for the absolute exclusion, by a Board of Special Inquiry, of idiots, imbeciles, feeble-minded, epileptic and insane, constitutional psychopathic inferiors and chronic alcoholic patients. The mentality of physical defectives required certification. The law of exclusion held without appeal for all classes of defectives except the physical, on which a board of special inquiry passes. The individual may appeal from the decision of this board, in which case the question is passed on to the Secretary of Labor who may grant admission under bond.

The medical work consists in recognizing the undesirables. The general procedure is as follows: The immigrants pass before the examiners and medical inspectors in line. At the head of the line stands a physician examining the eyes to make sure that no cases of trachoma pass. From the survey of immigrants as they pass, a rough general examination is completed, and persons presenting any abnormality are marked with chalk, the letters to designate the part affected, such as eyes, lameness, hernia, etc. The patient marked is then further examined. The one marked "L" for lame, for example, may show spastic paraplegia or locomotor ataxia, or hemiplegia, or progressive muscular dystrophy or multiple neuritis, etc. The examiner is called on to differentiate between countless causes. It is also a usual practice in making the routine examination to speak a word or so to each person and thus elicit a reply and in this way investigate speech. A total examination gives further information in regard to muscular atrophies, etc. It is then the duty of the medical officer to certify the neuropsychiatric conditions found. Sometimes the examiners differ. In cases of doubtful diagnosis the person is usually certified as having organic disease of the central nervous system.

The difficulty of arriving at a diagnosis is great. The immigrant often will not give any information, or if he does, it is intended to delude. The same difficulty is encountered in examining mental defectives and psychotic patients. Here the Binet-Simon test cannot be relied on because of the variety of standards and environment represented by these aliens. For instance, some immigrants have never seen electric light, they have no knowledge of dates, the number of months in the year, the days of the week, etc. The conditions imposed by foreign languages is also exceedingly difficult. Interpreters miss the points of interest and value to physicians. In a psychotic case, for example, the physician wants to know just what the patient says. The interpreter tries to make sense out of it and thus slurs the abnormalities in the current of thought.

Where a conclusion cannot be reached immediately the alien is sent to the hospital for observation. Thirty years ago there were no observation facilities. Now there is complete equipment and laboratories. In many cases, such as trachoma, mental deficiencies due to hypothyroidism or athyroidism and other conditions arising from disturbances of internal secretion, a diagnosis is not made until the result of treatment is seen. In other cases the diagnosis is not made until confirmed by bacteriologic examinations, as in leprosy, favus, ringworm, etc. Weekly conferences are held at which all interesting cases are presented and discussed and outside consultants come to lecture, etc.

DISCUSSION

Dr. H. V. Wildman, Jr., said that with the great influx of peoples from southern and eastern Europe the problem of keeping out those with mental defect was becoming one of the greatest importance. The detection of feebleminded immigrants is a separate and distinct specialty. Suggestions have been made that the Army B test be applied to arriving aliens, but experimental work with group tests in general and the B tests in particular have not given good results. The largest number of mentally defective aliens have been detected by a short personal interview with the individual alien during the line inspection.

A discouraging feature of the work at present is that many persons mandatorially excludable, are landed for various periods of time at the discretion of the Secretary of Labor. Lack of proper supervision allows some of these persons, through various contingencies to land permanently in the country.

Dr. I. J. Sands said that many of the immigrants who became ill might later be found in Bellevue. Many of those who were admitted to the observation wards echoed many of the horrors that had been occasioned by the late war. Thus, a Russian developed a psychosis while on the steamer on getting white bread to eat; he had been living on straw for several months, and the sight of real bread caused him to react in a manic-depressive fashion. Another case was that of a Russian Jewish girl who had thrown her brother's children from a window. She was in a state of depression when she came to this country, and after six months a manic state developed. Her original depression was undoubtedly occasioned by the hardships encountered abroad and by the difficulties met with while traveling through Russia and Poland. Dr. Sands thought that those immigrants that might later suffer from a neuropsychiatric condition to the extent of making them public charges, could hardly be detected by means of the ordinary so-called line examination. Cases of multiple sclerosis, paresis, etc., would hardly reach the boat; while cases of high grade mental deficiency, manic depressive phychosis in the mild and early stages and simple praecoxes, might easily escape detection. With an increase in the number of examining physicians, and the assignment of those men who have a knowledge of several of the foreign languages to Ellis Island, the number of undesirable aliens admitted would be materially reduced.

Dr. H. V. Wildman said that the percentage of mental defects picked up at Ellis Island was three times that of the feebleminded in institutions of the state of New York. Certificates of mental defects are issued only in cases about which there is no doubt.

In regard to the psychoses, the country is protected by the law which provides for deportation of aliens who become insane within five years of landing. A similar provision for cases of mental defects detected within five years after landing would seem an additional and desirable safeguard if it could be incorporated in the next immigration law.

Dr. Onuf, in closing the discussion, admitted that the examination could be made more thorough if the examining board had more men. The force is sometimes inadequate. When immigration is heavy fewer certifications are issued since the examiners have not time enough. Yet in spite of the rush a good number of psychoses are certified.

Book Reviews

LEITFADEN DER ELECTRODIAGNOSTIK UND ELEKTROTHERAPIE FUER PRAKTIKER UND STUDIERENDE. By PROF. DR. TOBY COHN, Neurologist in Berlin. Sixth Edition. Price 30 M. Pp. 226. Berlin: S. Karger, Karlstrasse 15, 1920.

The first edition of this work was published in 1898. It is apparent that the author has kept in close touch with all advances along physiologic, experimental, diagnostic and therapeutic lines. The book differs from the conventional presentations of this subject in being readable and at the same time sufficiently complete to serve as a reference work. It is divided into two parts; Part I takes up electrodiagnosis, Part II electrotherapy. There are numerous footnotes and insertions that are particularly interesting and instructive.

The physical characteristics of the electric current and the various types which find application in medicine are discussed in Chapter I. The second chapter deals with the physiologic problems that are involved. The methods of examination are in a general way sufficiently understood and need no further comment; a number of valuable technical hints, however, are given to overcome the difficulties that are ordinarily left for the tyro to discover and contend with.

In the discussion of various types of degeneration and changes in reaction that are found in muscles and nerves, the book conveys an assurance that the writer has had vast experience along these lines, since he also confesses to having difficulties in interpretation and departs from the tiresome orthodoxy that results in so much discouragement when one attempts to put what one reads into practice. In spite of this, unity and coherence are retained throughout in a most satisfactory way.

The electrical examination of sensory organs, such as the eye, the cochlear and the vestibular apparatus, is discussed but is not stressed as being very practical. This also applies to the discussion on the examination of cutaneous sensibility. The presentation of resistances is enlightening in that a subject is taken up that is usually neglected.

Part II is devoted to electrotherapy and dispels any lurking preconception concerning the writer's over enthusiasm. While he takes up the various methods that are employed in electrotherapeutics, he is always cognizant of the psychic factors. The physical action of the current on which much of the benefit from treatment depends is based on wandering of ions, thermic reactions, chemical reactions, ionphoresis, and some bactericidal properties; the physiologic action is dependent on vasomotor effects, changes in electrotonus, which is local rather than general and of limited value, contraction of the smooth and striated muscles, cutaneous reactions, and secondarily, effects on blood pressure and metabolism. Treatment of various diseases of the peripheral nerves, muscles, spinal cord and brain is taken up somewhat in detail. The treatment of the neuroses is considered rather briefly, probably because results depend almost entirely on psychotherapeutic factors of which electrotherapy should be only a small part. On the whole, the treatment of internal organs by such methods as necessitate the insertion of various electrodes is condemned, since the benefit that is derived is often questionable and the harm that may be done, considerable. On account of the effects on the mucous membrane incident to polarization, the use of the galvanic current is discouraged altogether.

Various types of apparatus, their construction and different advantages are dealt with. The chapter on franklinization is brief and quite in line with the discarding of the imposing influence machines from the modern office equipment, since no particular value is found here that cannot be obtained from other methods of obtaining the current. Arsonvalization, introduced by Tesla, is regarded with skepticism. The sinusoidal current is strongly condemned, since deaths have followed its application. Treatment with the Leduce current and with electromagnetism is briefly explained and discussed.

In the last chapter the condensor apparatus of Zanietowski is described. While comparatively little is known of it, the writer believes that examination by this method will become increasingly important, since it permits an accurate measurement, in microfarads, of the amount of electricity discharged. It has the advantages of being little influenced by cutaneous resistances and having a constant value as compared with those of the ordinary galvanic current as shown in Stinsing's tables.

THE BASIS OF PSYCHIATRY (PSYCHOBIOLOGICAL MEDICINE).

A Guide to the Study of Mental Disorders for Students and Practitioners. By Albert C. Buckley, M.D., Associate Professor of Psychiatry, Graduate School of Medicine, University of Pennsylvania. Cloth. Price, $7. Pp. 447, with 79 illustrations. Philadelphia: J. B. Lippincott Company, 1920.

Here we have a book of major length written from one courageously held point of view. Recoverable psychoses are regarded by the author as "reflections of some bodily disorder which through its effect on the organ of adjustment prevents the patient from making appropriate adaptation to environmental conditions." It follows that interest must be centered on those essential "bodily disorders" and "the organ of adjustment."

So we have a long and remarkable introduction in the first seven chapters. Attention is characteristically called to things outside of the skull, to the field of internal medicine, to muscles and glands which are meeting, or falling short of, or exceeding the demands made on them by a changing environment. A description of the development of the nervous system leads from Gaskill and Parker to Sherrington. In discussing salivary and gastric secretion, the author's goal is shown by his quotation from Pawlow: psychic activity "is nothing but an endless chain of reflexes." The conditioned reflex and the autonomic nervous system are again given the honor of providing the bridges to lead from laboratory to clinical observations.

Central localization and the anatomy of the brain in general are carefully written and proportioned to the needs of the reader of a textbook on psychiatry. A section on heredity is well illustrated and diagramed.

Mental development and psychologic processes are given nearly fifty pages, with at first comparisons with animal development and later much reference to the visceral accompaniments of mental states. The "unconscious" is discussed in the last four pages and with it freudianism in general.

No more interesting discussion of etiology is needed than that found in chapters 6 and 7, the minimizing of psychic causes making the handling of the problem more striking. Mental patients fall into two great classes:

"1. Those who develop psychoses irrespective of environment; for example, paranoia and manic-depressive psychoses. 2. Those who develop psychoses provided there exist the proper environment and exciting causes; for example, puerperal and other toxic psychoses, general paralysis and organic dementias —*accidental* psychoses." How many mental diseases may, after the fashion of general paralysis, develop years after a "mild" infectious process? The author thinks more in terms of widespread toxins than of focal infections, stressing disturbances of internal secretions, general effects of syphilis and tuberculosis, and in particular citing the importance of alcohol in mental disorders "produced" by head injury.

The chapters on classification, symptomatology and methods of examination are not unusual. Weston has called attention to some errors and omissions in the section on laboratory technic, and a manual other than this general work should naturally guide the laboratory worker.

The different psychoses are treated under nine headings in the second half of the book. All are defined in subheadings as reaction types due to infection, toxin, or degenerative brain process, or to unknown cause. The dementia praecox group appears "during the evolutive period, arising out of an inherent biogenetic defect, terminating frequently in neuronic degeneration, and characterized by an interference with the adaptive mechanism through inefficiency of the sensory, emotional, and volitional processes." The manic-depressive group is considered as a quantitative disturbance of everyday reactions. The psychoneuroses are "generally regarded as of psychogenic origin." The attractive chapter on "Psychoses with Somatic Diseases," is abstracted in the following paragraph as typical of the author's method:

These are mental "reactions dependent on functional disturbances of the organism due to" the inseparable trio "infection, exhaustion or toxemia, affecting the sensory motor and associating mechanisms, characterized by delirium, confusion, and physical prostration." They greatly concern the general practitioner who may meet delirium before, during and after a fever. Mental states are found which vary from insomnia and restlessness with depression through hallucinosis, confusion to wild excitements or long neurasthenias. On the physical side may appear twitching of muscles, nausea, constipation, dry or clammy skin and paresthesias. The pathologic condition consists of engorgement of membranes, disintegration in cortical neurons and cells of basal nuclei or hemorrhagic extravasations into the brain substance. In treatment we try to help an organism which not only cannot cope with the environment, but which is also poisoning itself, and needs a careful physical examination and a nurse trained in general medicine and psychiatry. Liquid diet is given in moderate amounts frequently with much attention to water supply, baths, and administration of alkalies. The minute directions should be carefully followed by any general physician who has to treat these infective-exhaustive conditions—which have a high mortality because in general their treatment is not understood.

In brief, this is a book worth owning, unusual in point of view and broad in scope, not attempting to cover the field of "psychic mechanisms," but emphasizing the broad foundation of psychiatry and many practical and exceedingly good points in treatment.

TRAVAUX NEUROLOGIQUES DE GUERRE (PAPERS ON WAR NEUROLOGY). By Georges Guillan and J. A. Barré. Paper. Price, 18 francs net. Pp. 460, with illustrations. Paris: Masson et Cie, 1920.

Introduction: This book of 460 pages is made up of a preface by Pierre Marie and six chapters:

Chapter 1, Neurological Symptomatology (fifteen papers); chapter 2, Wounds of the Brain (eight papers); chapter 3, Wounds of the Spinal Cord (seven papers); chapter 4, Concussion Without External Wounds Resulting from the Explosion of Shells (twelve papers); chapter 5, Pathology of the Cranial and Spinal Nerves (eight papers); chapter 6, Varia (five papers).

The first chapter is concerned with reflexes normal and abnormal. The second chapter is more interesting. The papers on a thalamic syndrome, on hemorrhagic meningitis without injury of the dura and on the late results of trephined cases are among the best.

The chapter on spinal cord wounds is full of interesting papers. That dealing with complete transverse lesions showed, among other things, that the deep reflexes were absent (Bastian's law) in thirteen out of fifteen cases. The reaction to plantar stimulation was present in fourteen out of fifteen cases, but was flexor in type. The other case showed no reaction. Other interesting observations on reflexes, both somatic and visceral, are to be found in this paper.

The paper on organic paraplegia from wounds of the vertebrae without dural rupture and that on lesions of the cauda equina contain much valuable material.

The chapter dealing with organic lesions after shell explosions which cause no wounds is too lengthy and detailed (eighty papers) for a review. It takes up the various aspects of this important subject quite thoroughly.

The next chapter contains papers on oculomotor paralysis during paratyphoid, ocular paralysis from picric acid poisoning with demonstrations of picramic acid in the spinal fluid and various unusual types of spinal nerve paralysis.

The last chapter deals with various subjects: spinal fluid hypertension following ligation of the jugular vein, periodic paralysis (Westphal-Goldflam), Landry's paralysis following antityphoid vaccination, radiculitis and gas poisoning.

The book gives a good idea of war accidents involving the central nervous system. The subject of involvement of peripheral nerves is not brought up except in a few case reports. The book is worth reading, is well written and well illustrated, and has a variety of material which should be of interest to both the medical and surgical neurologist.

THE CROONIAN LECTURES ON THE PSYCHOLOGY OF THE SPECIAL SENSES AND THEIR FUNCTIONAL DISORDERS. By Arthur F. Hurst, M.A., M.D., Oxon, F.R.C.P., Physician and Neurologist to Guy's Hospital. Pp. 122. London: Henry Frowde, Hodder & Stoughton, 1920.

After carefully reading this work, one is tempted to say "a little book with a big I." Essentially it is clinical, a report of cases of hysteria (mostly in soldiers) seen by the author. Of psychology, either of the special senses or of hysteria, there is just a trace. What strikes the reader is the constant emphasis placed on the discoveries and opinions of the author. The former are almost entirely rediscoveries of things known for many years or familiar to all neurologists who have done war work or read of it. The latter are for the most part either tiresomely trite or not those of a mature neurologist.

The psychology, what there is of it, is naive to a degree—quite "childlike and bland." Briefly stated, it is this: The person hysterically blind is so because he does not look, the hysterically deaf is so because he does not listen and the hysterically anesthetic does not feel because he does not pay attention. The mechanism is that the lack of attention and failing to look and listen cause a break in the sensory afferent tract somewhere in the brain stem by retraction of dendrites at a synapse.

But if the book is neurologically and psychologically weak, not to say amateurish, and the accomplishments of the author exalted to an unwarranted degree, it is still a very readable and interesting record. Hysteria, the protean, is always interesting, and if the author does not greatly edify us, his cases do. Some of his own work is also interesting. "Twenty-nine healthy and intelligent individuals, of whom twenty-five were medical students, who had, however, not yet worked in the wards or read any clinical medicine, were asked to pretend that they had been in a railway accident, and that they were attempting to swindle the company by claiming compensation because of paralysis of the right arm and leg, which they alleged had resulted. They were to act as well as they could the part of men trying to persuade the medical examiner that they were really suffering from paralysis." The results were quite comparable to the symptoms of traumatic hysteria.

The therapeutic procedures employed are along the lines of persuasion and suggestion with which all neurologists are familiar, and which are good. But the author falls into the old error of post hoc propter hoc. Because his theory of hysterical deafness when explained to the patient helps to cure him, the author concludes that it is the real explanation of the deafness. When a magnet transfers anesthesia or banishes paralysis we do not conclude that there is virtue in the magnet, but that the patient has accepted a helpful idea. That Hurst's conception of hysterical blindness and deafness may appeal to and be accepted by an intelligent person, is shown by its acceptance by Hurst himself.

As a clinical assistant the book will be very useful to those not familiar with hysteria, and for all it is interesting and stimulating.

A STUDY OF WOMEN DELINQUENTS IN NEW YORK STATE.

Mabel Ruth Fernald, Mary Holmes Stevens Hayes and Almena Dawley, with a statistical chapter by Beardsley Ruml, and preface by Katherine Bement Davis. Pp. 542. New York: The Century Company for the Bureau of Social Hygiene.

The purpose of this comprehensive study was to attain a more adequate understanding of women delinquents. The groups of women studied include 102 at Bedford Reformatory; 88 at Auburn State Prison; 76 at the New York Magdalen Home; 110 at the New York County Penitentiary; 109 at the New York City Workhouse and 102 probation cases from the women's night court of Manhattan and the Bronx, making a total of 587 cases. The results are compared with those found by other investigators both in groups of delinquents and of general population. The principal topics treated are: nature and extent of delinquency; age; habits with respect to use of tobacco, alcohol and drugs; general social factors; nativity and color; early home conditions and family status; educational background, occupational, history and economic efficiency; sex irregularities and mental capacity.

In one of the most valuable chapters in the book mental capacity is correlated with other factors which might influence delinquency. The authors find that the average mental capacity of women delinquents is somewhat lower than that of the average of women in the general population. They do not, however, agree with Goring that constitutional factors are of greater importance than environmental factors as determinants of crime. The fact is emphasized that delinquency in women cannot be traced to any one predominant factor but results from a large variety of interrelated causes.

The study seems to indicate that native born colored women have the highest rate of delinquency, native born white women a considerably lower rate and foreign born white women a still lower rate.

As a whole, this is a valuable contribution to our knowledge of women delinquents and great credit is due the authors for their thorough investigation of individual cases. One could feel surer of the statistical conclusions if the number of cases studied were larger. The statistical tables and drafts are admirably presented, and the limitations of each are clearly set forth. The frequent use of standard deviations and ratios of correlations with the accompanying discussions of the probability of error make some of the discussions too technical for the average reader. This painstaking statistical work, however, increases the value of the book to the student of social science.

GRAPHIC METHODS IN HEART DISEASE. By JOHN HAY, M.D., F.R.C.P., Honorary Physician, Liverpool Royal Infirmary; Honorary Consulting Physician, Ministry of Pensions; Later Consulting Physician in Diseases of the Heart (Western Command); with an Introduction by SIR JAMES MACKENZIE, M.D., F.R.C.P. Pp. 172. London: Henry Frowde, Hodder & Stoughton, 1921.

This book is chiefly concerned with a discussion of the use of the polygraph in disease of the heart. The exposition of the subject is clear, concise and free from unnecessary and bewildering details. The mechanism of cardiac irregularities and the aid to be derived from the polygraph in their interpretation are made plain. The numerous figures, most of them original, are, because of the fine quality of paper employed, of unusual excellence. In brief, we know of no bettter discussion of the essentials of polygraphy than is given by Hay in this small volume.

Since the publication of the first edition twelve years ago, the electrocardiograph has largely supplanted the polygraph. Some even go so far as to say that the polygraph is now of historical interest only. But because of its relative cheapness and its portability, the polygraph still remains the more serviceable instrument for the practitioner. Hay has added a brief sketch of electrocardiography with several typical electrocardiograms that will give the reader a glimpse of what may be revealed by this wonderful machine.

The question is often asked: Of what practical use are the polygraph and the electrocardiograph? The last page of the book contains the best answer to this question we have ever seen. The answer, as given by Hay, is that these instruments "create a new interest." He expounds this text in a few short but illuminating sentences.

TROPICAL OPHTHALMOLOGY. By Robert Henry Elliot, M.D., B.S. (Lond.), Sc.D. (Edin.), F.R.C.S. (Eng.), Lieutenant-Colonel, I. M. S. (Retired), Late Superintendent of the Government Ophthalmic Hospital, Madras and Professor of Ophthalmology, Medical College, Madras; Honorary Fellow and Gold Medallist of the American Academy of Ophthalmology and Otolaryngology; Lecturer in Ophthalmology, London School of Tropical Medicine; Ophthalmic Surgeon to the Seamen's Hospital Society, and to the Hospital for Tropical Diseases, Endsleigh Gardens, London. Pp. 502, with 7 plates and 117 illustrations. London: Henry Frowde, Oxford University Press, Hodder and Stoughton, Warwick Square, E. C., 1920.

This work is practical and readable, the fruit of about twenty years of experience in India. The author shows that tropical ophthalmology has become a special study, as much from the habits of mind and body of the natives as from the influences of a tropical climate. For example, we are confronted with a prophylactic operation for glaucoma. Besides a comprehensive discussion of the various tropical parasitic diseases of the eye, the author gives careful attention to the more unusual external diseases, such as rare forms of keratitis, Herbert's corneal plaques, conjunctival granuloma, and other conditions. An interesting and instructive account is given of the native operations of couching for cataract, in which one makes a capsulotomy with a needle knife before making the corneal section. The very short chapter on ophthalmoscopic appearances in the tropics (by Lieut.-Col. H. Kirkpatrick) seems all too short, and it is to be hoped that a subsequent edition may devote more attention to this branch. The author's discussion of eye conditions in general diseases in collaboration with Professor Miyashita of Japan is very full. The book is generously illustrated with photographs, original drawings and excellent colored plates.

UEBER DIE JUVENILE PARALYSE. Toni Schmidt-Kraepelin, Berlin, Julius Springer. Pp. 124. 1920.

In this monograph is presented the first part of a consideration of congenital syphilis in its relations to mental abnormality, which is finally to include the neurologic disturbances of idiocy and imbecility, epilepsy, syphilis of the brain, and even psychoneurotic tendencies in the apparently sound offspring of syphilitic patients.

Dr. Kraepelin presents only juvenile general paralysis, in thirty-nine cases of pure type, seven doubtful cases, and seven cases in which serologic, but not clinical, positive findings (pre-paralysis?) were obtained. She makes a thorough analysis of histories, symptoms and events in the course of the disease processes. Thirty-seven tables explain this part of the text alone, including tables showing metasyphilis in parents, Wassermann reactions in relatives, the preliminary symptoms, neurologic signs and treatment. A section follows which gives the individual records in painstaking detail with photographs in full length of the pure types. Consideration of the doubtful cases is clarified by more tables (up to Table 53), including a comparison of constitutional traits, stigmas, neurologic, physical and mental symptoms.

The conclusions are in general not unexpected. When one adds the frequency of focal symptoms, ataxias, pseudo-choreas, and infantilism to a known hereditary cause, it becomes more than ever necessary to rule out general paralysis when diagnosing the neurologic diseases of children.

KURSUS DER PSYCHOTHERAPIE UND DES HYPNOTISMUS (A COURSE OF PSYCHOTHERAPY AND HYPNOTISM). Von San.-Rat. Dr. Georg Flatau, Nervenarzt in Berlin. Zweite durchgesehene Auflage. Pp. 175. Berlin: Verlag von S. Karger, Karlstrasse 15, 1920.

This is a manual not of psychotherapy, but of hypnotism for the physician, with just enough psychology to make it seem scientific and reasonable. When the historical part, general considerations and theories are left out, there remains a fair exposition of the methods, practice and results of a physician who constantly uses hypnotism. His technic is given and the reasons for it (and they are reasonable), the disorders in which he habitually uses hypnotism and comparison of this method with "persuasion." Incidentally, the author states that he has cured patients who had not been relieved by psychanalysis.

It is pleasant to note that the author insists that the operator must be a good physician first and a hypnotist afterward.

Any physician wishing to brush up on this therapeutic procedure will find the book satisfactory.

CLINICAL OPHTHALMOLOGY FOR THE GENERAL PRACTITIONER. A. Maitland Ramsay, M.D., Fellow of Royal Faculty of Physicians and Surgeons, Glasgow; Lecturer on Ophthalmology, University of Glasgow; Ophthalmic Surgeon, Glasgow Royal Infirmary; Author of "Atlas of External Diseases of the Eye," "Eye Injuries and Their Treatment," "Diathesis and Ocular Diseases," etc. With Foreword by Sir James Mackenzie, M. D., F.R.S. Pp. 476. London: Henry Frowde, Oxford University Press; Hodder & Stoughton, Warwick Square, E. C. 4, 1920.

In this book the author has abandoned the usual systematic presentation of particular diseases, adopting a less conventional, more discussive method, with the object of better meeting the needs "of the busy country doctor." For instance, instead of a chapter on glaucoma we find one on "The Clinical Significance of a Hard Eye." There may be advantages in speaking of the glaucomatous eye simply as a hard eye, but the lack of system is shown in considering first diagnosis and then indirectly etiology and pathology. For the general practitioner full consideration of the eye in relation to general disease would seem to be desirable but this has hardly been done in the brief treatment given choked disk, neuroretinitis, optic atrophy and arteriosclerosis. One is further disappointed in the lack of adequate attention given to the general factors in choroiditis and retinitis. The author is not wisely consistent in his advice to the general practitioner as to what to do and what not to do. He gives elaborate and minute details of procedure in the operation of iridectomy for glaucoma, but does not once say this operation should be done only by a skilled operator. On the other hand, he advises the practitioner not to do ectropion and entropion lid operations, which he might well perform.

On the whole, one must say that while the book is interestingly written and well illustrated with cuts and colored plates, and the busy physician could glean considerable useful information from it, a smaller book, with the subject matter arranged in a more scientific and definite manner, would better meet the needs of the general practitioner.

SURGICAL ASPECTS OF DYSENTERY, INCLUDING LIVER ABSCESS.

By Zachary Cope, B.S., M.D., M.S. (Lond.), F.R.C.S. (Eng.), Surgeon to Outpatients St. Mary's Hospital; Surgeon to Balingbroke Hospital; Captain, R. A. M. C.; T. F. Surgical Specialist with Mesopotamian Expeditionary Force, 1916-1919; Hunterian Professor R.C.S. Cloth. Pp. 144. Oxford Medical Publications.

This work is based on the personal experience of the author and his associates in the Royal Army Medical Corps with the surgical complications arising from approximately 2,000 cases of dysentery, of both amebic and bacillary type. The subject is considered from three standpoints: (1) dysentery as a complication of surgical conditions, (2) various surgical conditions simulated by dysentery, and (3) surgical complications in the course of dysentery. He emphasizes especially the value of appendicostomy, cecostomy, or cecostomy with complete enterostomy in chronic unyielding cases, and warns against too great delay in operating. Surgeons operating on liver abscess are urged to keep a keen lookout for cerebral symptoms. He states that in dysentery abscess of the brain never occurs apart from liver abscess but that, according to Kartulis and Jacob, it occurs in 3 per cent. of liver abscess cases.

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ANATOMIC AND PHYSIOLOGIC STUDIES OF THE EIGHTH NERVE

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INTRODUCTION.
REVIEW OF LITERATURE.
METHODS USED IN INVESTIGATION.
PURPOSE OF INVESTIGATION.
CONNECTIONS OF SEMICIRCULAR CANALS WITH MEDULLA.
SYMPTOMS ARISING FROM DESTRUCTION OF SEMICIRCULAR CANALS.
ANATOMIC CONNECTIONS OF VESTIBULAR AND ACOUSTIC BRANCHES OF EIGHTH NERVE IN MEDULLA, PONS AND CEREBELLUM.
SYMPTOMS RESULTING FROM CUTTING EIGHTH NERVE.
SYMPTOMS ARISING FROM BONE WAX TUMORS.
CONCLUSIONS.

Although many investigations have been conducted on the eighth nerve, there still remain numerous questions both as to the anatomy and physiology that have not been solved or about which there is some difference of opinion. In the last few years the interesting clinical observations of Bárány and Neumann [1] in Vienna, and Jones and Mills [2] and others in this country, have made the subject a particularly enticing field for further studies. Both the European and American observers have deduced anatomic and physiologic facts from their clinical observations. In some cases they have assumed the existence of anatomic pathways in order to explain adequately their clinical findings. The Bárány enthusiasts have gone so far that many of them believe to be facts what a few years ago were only theories. The purpose of the present investigation has been to study by modern and somewhat different methods the eighth nerve mechanism with special reference to the vestibular portion.

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[1] Bárány, R.: Ueber dem vom Ohre auslösbaren Nystagmus Vortrag a. d. 77, Vers. deutsch, Naturforsch. u. Aerzte Ztschr. f. Ohrenheilk. **51:** No. 1. Vestibular Erkrankungen und Neurose. Vortrag. geh. in Neurologische Gesellschaft zu Wien, Jan. 9, 1906, Wien. klin. Wchnschr. 1906, p. 489. Untersuchungen über den vom Vestibular Apparat des Ohres reflectorisch ausgelösten rhythmischen Nystagmus und seine begleitende Erscheinungen. Monatschr. f. Ohrenheilk. **11:**193-297, 1906. See also Ebenda, 1907. Beitrag zur Lehre von den Funktionen der Bogengänge, Ztschr. f. Psychol. u. Physiol. d. Sinnes Organen **41:** Pt. 2, 37-44, 1906. Ueber die vom Ohrlabyrinth ausgelöste Gegenrollung der Augen bei Normalhörenden, Ohrenkranker und Taubstummen, Arch. f. Ohrenheilk. **68:**1-30, 1906.

[2] Mills and Jones: Neuraxial Differences of Fibres from Horizontal and Vertical Canals Determined by Bárány Tests, J. Nerv. & Ment. Dis. **44:**245, 1916.

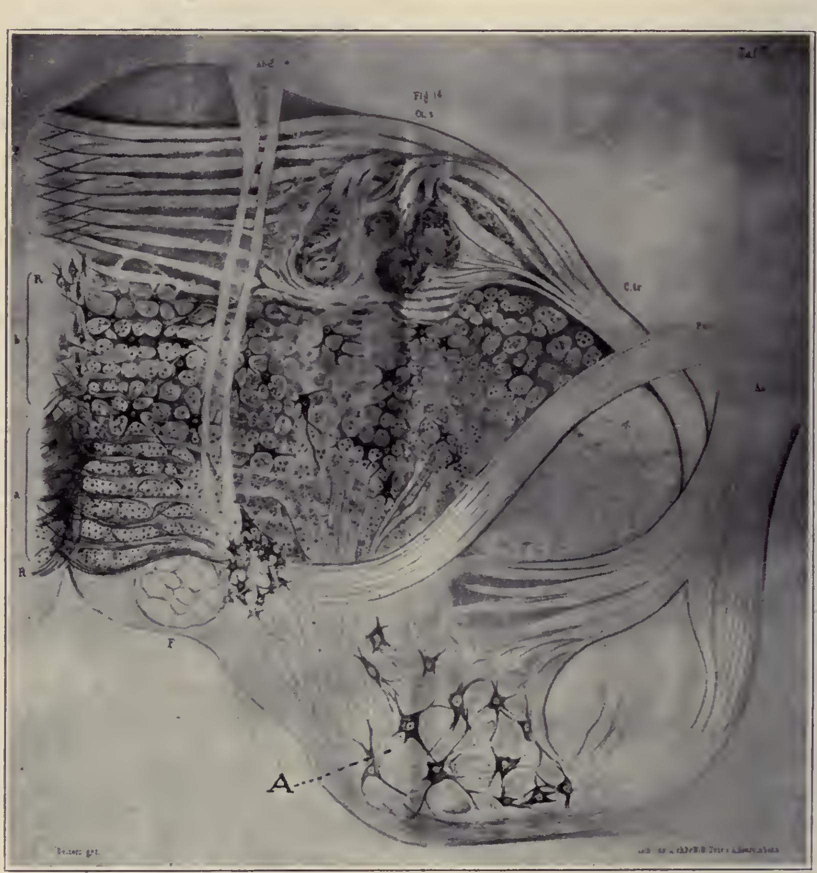

Fig. 1.—Illustration from Schultze's book on Deiters' work. This is Deiters' original drawing and shows that what is now called von Bechterew's nucleus is the nucleus Deiters originally described, and which Schultze named Deiters' nucleus.

REVIEW OF LITERATURE

One part of the physiologic phase of the subject has been completely reviewed up to 1894 by von Stein [3] in his monumental book of almost 700 pages. Most of the book, however, is devoted to the vast amount of work that has been done on the cochlea and semicircular canals. The book contains little mention of the work on the central connections of the eighth nerve.

As our studies have been along anatomic and physiologic lines, we shall not review the work of Bárány, Neumann, Jones [4] and those others who have dealt with the eighth nerve clinically. We shall briefly summarize the important points that have been described by previous observers which bear on this investigation. These articles will be taken up in chronologic order.

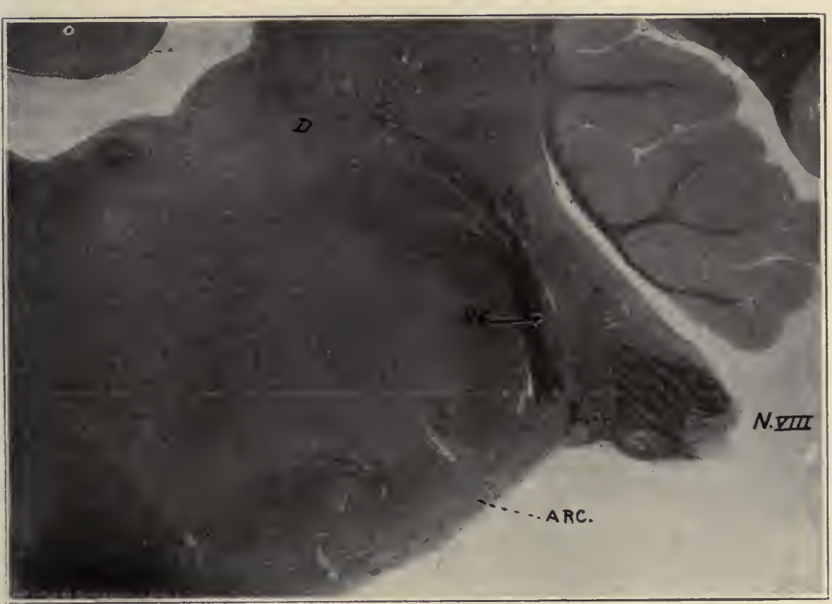

Fig. 2.—Experiment 135. The eighth nerve, *N VIII*, is completely cut across. Degenerated vestibular fibers, *VE,* are seen running to Deiters' nucleus, *D*. The arcuate fibers, *ARC*, are well seen. Note the slight injury to the adjacent cerebellum.

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[3] Von Stein, Stanislav: Die Lehren v. d. Functionen der einzelnen Theilen des Ohrlabyrinthes zusammegestellt von S. v. Stein, deutsch von Krzywicky. (Verlag G. Fischer, Jena).

[4] Jones, I. H. and Fisher, L.: Equilibrium and Vertigo, Philadelphia, Lippincott Co., 1918.

Von Monakow[5] (1882) made this statement: "An isolated extirpation of the eighth nerve for the purpose of studying the secondary atrophy is hardly feasible because of the impossibility of avoiding injury to neighboring parts."

Von Bechterew[6] (1885 and later) described the following symptoms when the posterior cerebellar peduncle was cut: "rolling over and over, circus movements in the long axis of the body." When the eighth nerve was cut on one side, the animal rolled to the side of the operation. When the region of the aqueduct of Sylvius or the anterolateral portion of the fourth ventricle was cut, the animal rolled to the opposite side. Nystagmus was present in all experiments. The animals lived only one or two days after operation. The specimens were studied by the Weigert method. There are no drawings or photographs to show the lesions. He concluded that the tracts from the olive and vestibular nerve to the cerebellum passed up the posterior peduncle,

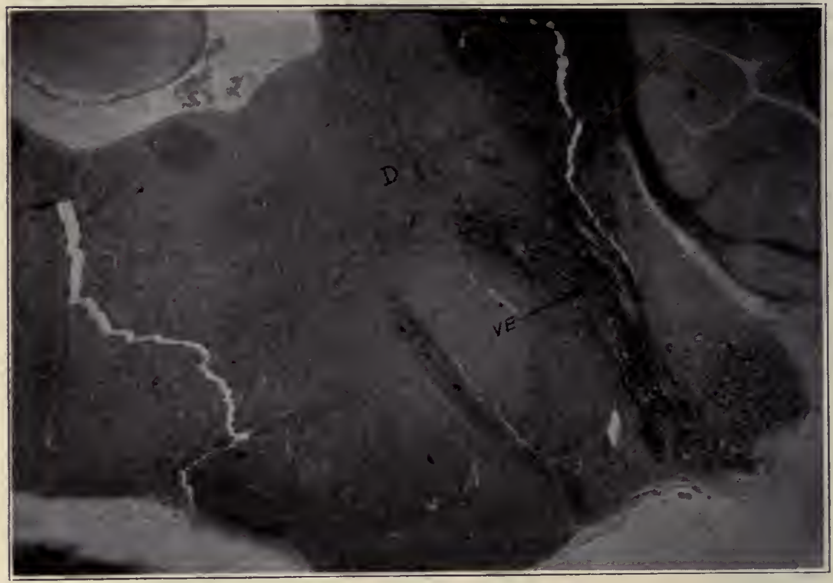

Fig. 3—Experiment 92. Vestibular fibers, *VE*, going to Deiters' nucleus, *D*.

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[5] Von Monakow: Experimentel Beitrag zur Kenntnis d. corp. Restiforme, Arch. f. Psychiat. **14**: No. 1.

[6] Von Bechterew: Ueber die Verbindungen der oberen Oliven und ihre wahrscheinliche physiologische Bedeutung, Neurol. Centralbl., 1885, No. 21. Zur Anatomie der Schenkel des Kleinhirns, ibid., p. 121. Zur Frage über d. Ursprung des Hörnerven über d. physiolog. Bedeutung des N. Vestib., ibid., 1887, p. 193

also that the olivary tract fibers decussate in the cerebellum. He was one of the first to work on the connections between the semicircular canals and the cerebellum.

Onufrowitsch,[7] working with Forel, studied the brains of two rabbits on whom at birth one otic bone had been destroyed. They studied the brains by noting the secondary atrophy and found that the vestibular fibers ended in the tuber acousticum.

Baginsky[8] (1885 and 1886) expressed the view that the symptoms von Bechterew described were due not to injury to the eighth nerve, but to lesions of the neighboring parts of the brain. In his own experiments he obtained the same symptoms and demonstrated lesions of the brain. In a later article he pointed out that only the posterior part of the eighth nerve is connected with the cochlea. Fibers from this portion pass to the tubercle and in part circle the corpus restiforme dorsalward. Many fibers also pass ventrally as arcuate fibers to the opposite side. He believed that only the anterior of the three acoustic

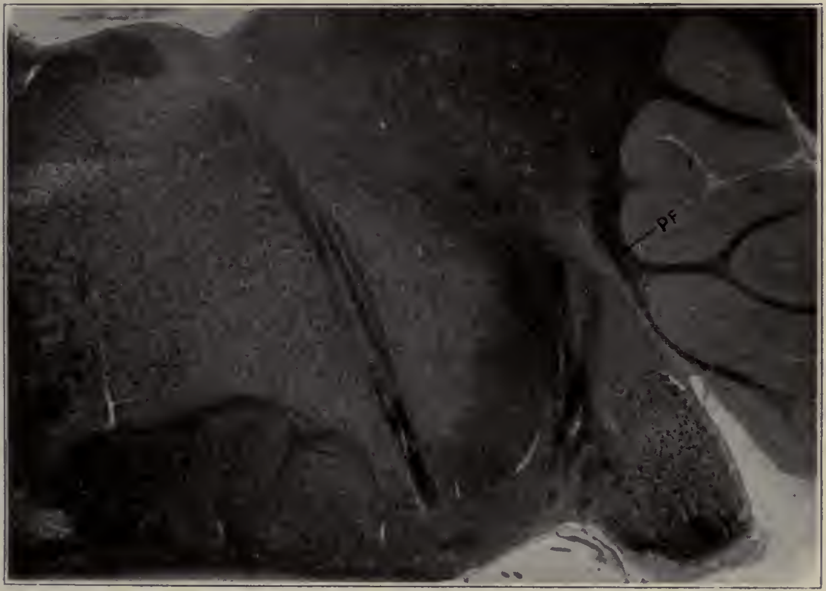

Fig. 4.—Vestibular fibers running to Deiters' nucleus. This illustration shows the amount of degenerated fibers, *PF,* resulting from retraction of the paraflocculus.

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[7] Onufrowitsch: Experimenteller Beitrag zur Kenntnis des Ursprungs des Nervus Acusticus, Arch. f. Psychiat. **16**:3.

[8] Baginsky: Zur physiologie du Bogengänge, Arch. f. Physiol., 1885, p. 252. Ueber d. Ursprung u. d. centralen Verlauf d. Nervus akust. d. Kaninchens. Sitzb. d. k. preussischen Akad. d. Wissenschaft **25**: (Feb.) 1886.

nuclei belonged to the posterior root. Fibers also go to the posterior corpus quadregeminum and internal geniculate body. There is no connection between the root and the cerebellum.

Bumm[9] (1889), working on rabbits at birth, believed that the posterior root of the eighth nerve had no connection with the cerebellum. The anterior acoustic root arises from the medulla and part of the cerebellum. Its origin is at the ventral border of Deiters' nucleus and in the vermis.

Biedl[10] (1895) used the Marchi method. His animals showed circus movements, rolling over and over and forced extension movements. Fibers pass to both posterior longitudinal fibers; others pass by the way of the arcuate fibers to the other side to form the lateral lemniscus; a third group pass to the olive of the opposite side and travel centrifugally.

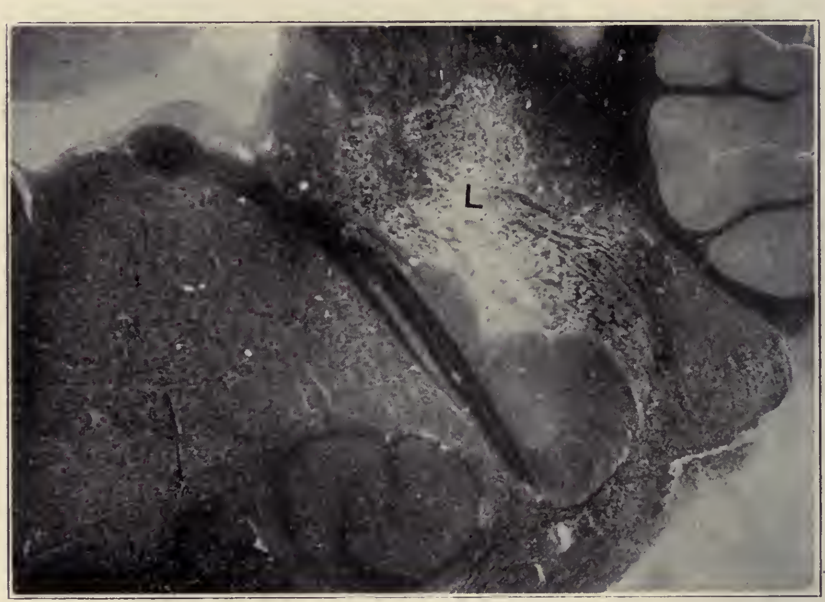

Fig. 5.—Experiment A5. Lesion, *L,* of Deiters' nucleus and Bechterew's nucleus.

Ferrier and Turner[11] (1894, 1895) sectioned the eighth nerve in monkeys. They observed rolling movements, an attitude of the head, extension of the limbs on the opposite side, but no nystagmus. The degenerations, as seen by the Marchi method, showed fibers (1) to Deiters' nucleus, (2) to the outer angle of the fourth ventricle, (3) arcuate fibers to the tegmentum of both sides, (4) to the superior cerebellar peduncle, and (5) to the sixth nucleus.

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[9] Bumm: Exp. Beitr. zur Kenntniss des Hörnervenursprungs beim Kaninchen, Allgem. Ztschr. f. Psychiat. **45**:568-572, 1889.

[10] Biedl: Absteigende Kleinhirn-bahnen, Neurol. Centralbl., 1895, p. 434.

[11] Ferrier and Turner: On Cerebro-Cortical Afferent and Efferent Tracts, Philadelphia Trans., B **190**:1-44, 1898.

Oseretzkowsky[12] (1895) made the same observations as the preceding observers, but he emphasized the fact that the arcuate fibers send many fibers to the superior olive of each side and that the decussating fibers from the cochlear nerve are more numerous than those on the same side.

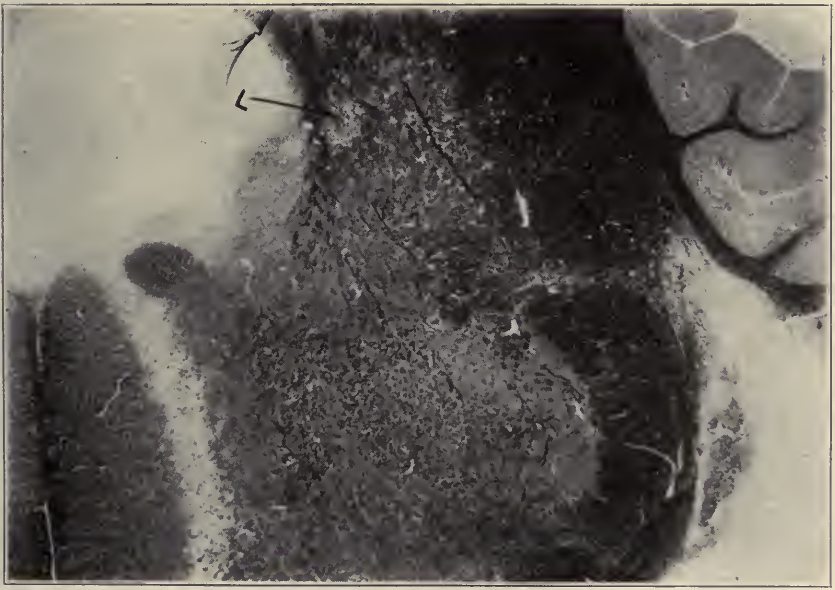

Fig. 6.—Experiment A5. Lesion, *L*, of Bechterew's nucleus.

Thomas[13] (1898) seems to have been the first to cut the eighth nerve intracranially on a dog. He made one experiment. He seems to have studied only the degenerations of the cochlear fibers.

Biehl[14] (1900) experimented on horses and sheep. The horses all died in from one to seven days. Some showed the so-called classical symptoms described by Bechterew. One of his sheep showed none of these symptoms. There was no histologic examination of these specimens.

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[12] Oseretzkowsky: Zur Frage vom centralen Verlaufe des Gehörnervs, Arch. f. mikr. Anat. **45**:450, 1895.

[13] Thomas: Les terminaisons centrales de la racine labyrinthique. Compt. rend. Soc. de biol., 1898, p. 183.

[14] Biehl, K.: Ueber die interkranielle Durchtrenung des N. vestibularis und deren Folgen, Sitzungsberichte der Wiener akad. math.-naturklasse **109**: Pt. 3, (July) 1900.

Fraser[15] (1901) made lesions of Deiters' nucleus and produced vestibular disturbances. He used the Marchi method and showed ascending and descending degenerations on the same and opposite sides. He found fibers going to the posterior longitudinal bundle, which he traced to the third and fourth nuclei and also followed fibers to the anterior corpus quadrigeminum.

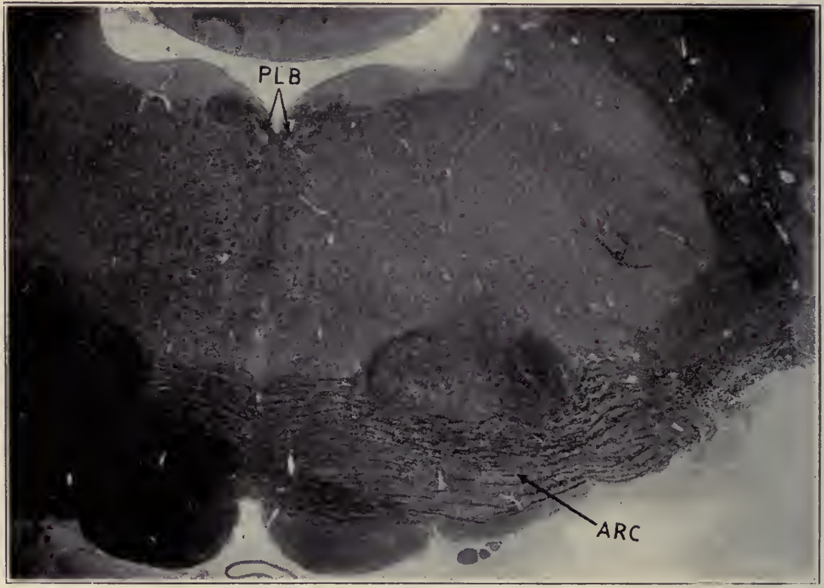

Fig. 7.—Experiment A5. Fibers going to posterior longitudinal bundles, *PLB,* of both sides; arcuate fibers, *ARC,* to superior olive.

Weigner[16] (1903), working on *Spermophilus citilus,* destroyed the cochlea in some cases with hydrochloric acid, in others with a blunt instrument. He used Marchi, Van Gieson and Weigert stains. His conclusions were:

1. Vestibular fibers pass through Deiters' nucleus.
2. The posterior acoustic nucleus is formed of small cells.
3. Fibers of the nucleus cochlearis pass to the nucleus ventralis and tuberculum acousticum and end there.

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[15] Fraser, E. H.: An Experimental Research into the Relations of the Posterior Longitudinal Bundle and Deiters' Nucleus, J. Physiol. **27**:372, 1901.

[16] Weigner: Exper. Beitrag. zur Frage v. zentralen Verlaufe d. Nervus Cochlearis bei Spermophilus citilus, Arch. f. mikr. Anat. **62**:1903.

4. From this gray mass fibers spring which pass dorsally and ventrally.

Van Gehuchten [17] (1903, 1904, 1905, 1906) observed for the most part what our work has confirmed. He asserts, however, that no fibers of the cochlear branch pass to the posterior corpus quadrigeminum; they stop before they get there. He is in accord with Fraser that the fibers in the posterior longitudinal bundle pass to the third and fourth nuclei. He believes that many of the degenerations observed by Tricomi-Allegra [18] (1905) are due to traumatism of other regions during operations.

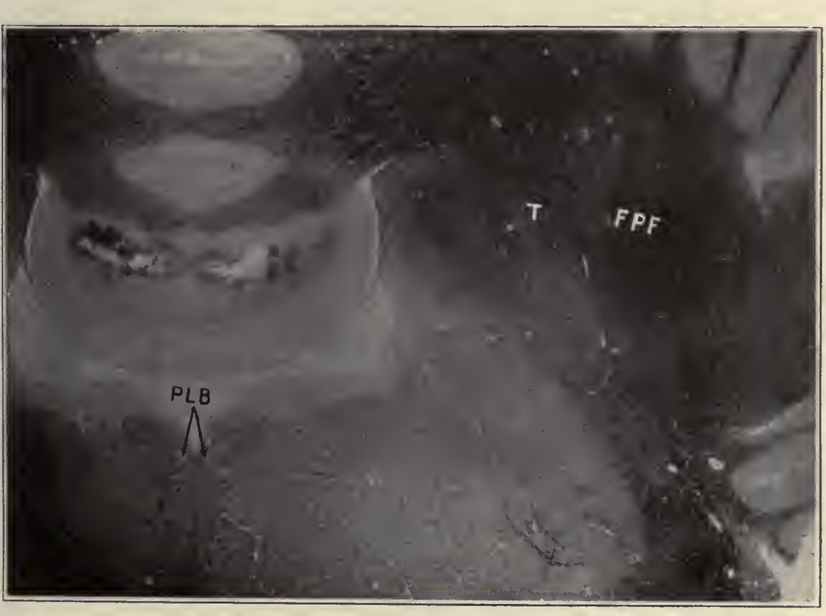

Fig. 8.—Experiment A5; same section as Figure 7. Fibers running into cerebellum; also fibers to posterior longitudinal bundle, *PLB*. Fibers, *FPF*, from injured paraflocculus can be seen going to lateral lobe and fibers, *T*, from vestibular nerve are seen going to nucleus tecti.

Matte [19] (1907) asserted that he found fibers which pass directly from the membranous labyrinth to the cerebellum.

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[17] Van Gehuchten: Recherches sur la voie acoustique centrale, Le nevraxe **4**: 1903. Connexions centrales du Noyau de Deiters et des masses grises voisines, ibid. **6**:21, 1904. La loi de Waller, ibid., 1905, p. 203. Le nerf cochleaire, ibid. **8**:127, 1906. Van Gehuchten and Molhaut: Les lois de la degenerescence wallérienne directe, ibid. **11**:73, 1910.

[18] Tricomi-Allegra: Studio sperimentale sulla via acustica fondamentale, Le nevraxe **7**:1905.

[19] Matte, F.: Ueber die Frage nach dem Auftreten von sekund. aufsteig. Degen. in. Stamme d. N. Acusticus nach Exstirpation einzelne Teile oder d. ganzen. Häutigen Ohrlab. Zentralbl. f. Physiol., 1907, p. 827.

Wilson and Pike[20] (1912-1913) believe that though the slow component of nystagmus is not affected by removal of the cerebellum or cerebrum, the quick component is affected by the removal of the cerebrum even though the oculovestibular tract is unimpaired. Whether the so-called oculovestibular tract was really unimpaired does not appear from their paper for they report no microscopic examination of their specimens, and with the very gross intracranial operations they performed as evidenced by the photographs of their specimens, it hardly seems possible that the oculovestibular tract could have escaped.

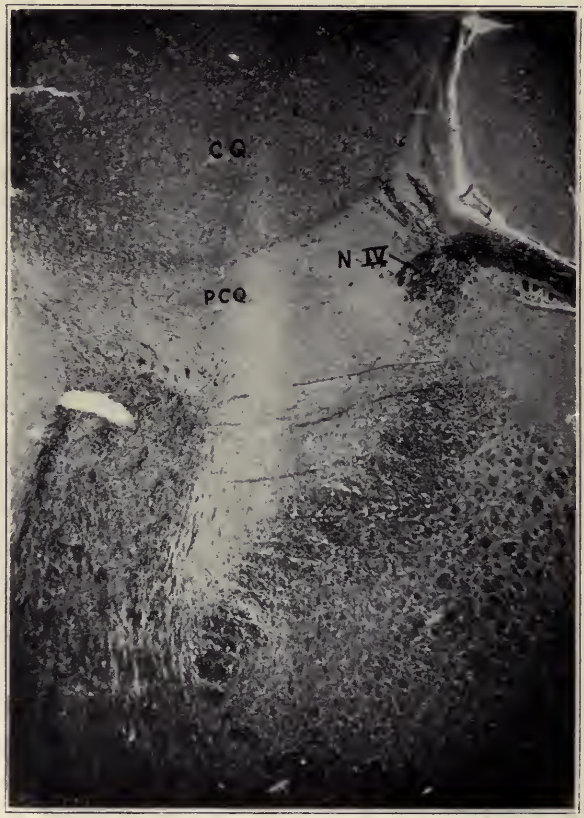

Fig. 9.—Experiment A5. Fibers, *PCQ*, running from lateral fillet into the posterior corpus quadrigeminum, *CQ*, of opposite side. These fibers come from the lesion in Deiters' nucleus, Figure 6. Nucleus of fourth nerve, *N IV;* no fibers from posterior longitudinal bundle running to it.

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[20] Wilson and Pike: The Effects of Stimulation and Extirpation of the Labyrinth of the Ear and Their Relation to the Motor System, Philadelphia Trans. Section B **203**:297, 1912.

Thus in their experiment, described on page 145 of their article and shown in Figure 18, it is impossible to assume that at least parts of the superficial fibers were not involved, yet the authors say "There could have been no irritation of the pons as this would tend to cause contraction of the pupil." This hardly seems a justifiable conclusion, for we have injured the pons a number of times without any effect on the pupil.

On the intracranial connections of the two portions of the eighth nerve there are also a large number of important papers. Of these, those by Van Gehuchten[17] and his pupils, Fraser,[15] von Bechterew,[21] Biehl,[14] Probst,[22] Ferrier and Turner,[23] Thomas,[13] Russel, and Sabine, are of greatest interest to us in this particular investigation.

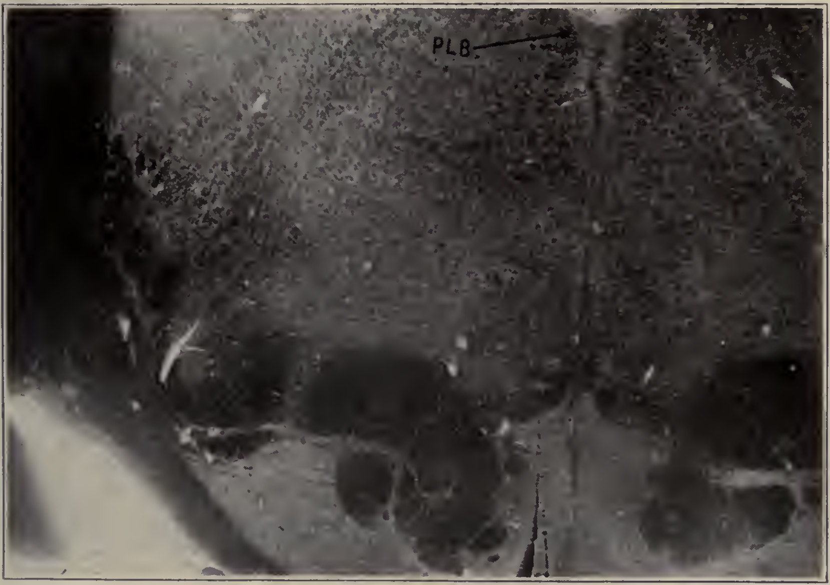

Fig. 10.—Experiment A5. The posterior longitudinal bundle, *PLB*, contains no degenerated fibers. This section is taken somewhat caudalward of fourth nucleus.

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[21] Von Bechterew: Ueber die Verbindung der sogenannten peripheren Gleichgewichtsorgane mit dem Kleinhirn, Pflüger's Arch. f. Physiol. **34**:352, 1884. Ergebnisse der Durchschneid des Acusticus, ibid. **30**:312. Footnote 6.

[22] Probst: Ueber vom Vierhügel, von der Brücke und vom Kleinhirn absteigende Bahnen, Deutsch. Ztschr. f. Nervenheilk. **15**:192-219, 1899. Experimentelle Untersuchungen über die Anatomie u. Physiologie der Leitungsbahnen des Gehirnstammes, Arch. f. Anat. u. Physiol. Anat. Abth., 1902.

[23] Ferrier and Turner: Experimental Lesions of the Cerebellum and Peduncles in Monkeys, Philadelphia Trans. Section B **185**. II. 1894; also footnote 10.

METHODS USED IN THIS INVESTIGATION

We have performed in all 109 experiments on dogs. Many of these, however, had to be discarded for a variety of reasons. A few were infected, others died from exhaustion due to thrashing about after operation as a result of their violent ataxia and circus movements. In a number of cases the staining was inadequate. This has reduced the cases on which these studies are based to sixty-nine. Some animals operated on with the idea of producing a certain lesion were subsequently transferred to another group after anatomic studies had been completed.

Of these sixty-nine, thirty experiments were made on the semicircular canals. The method described by Wilson and Pike [20] for exposure of the canals was used, but one of us (B. Y. A.) improved on it so that he was able to destroy one canal and its ampulla. This was done by opening the canal and introducing a fine wire and destroying the canal and its ampulla. In a few cases all three canals were destroyed by opening one canal and injecting 95 per cent. alcohol stained with eosin. The location of the eosin in the dissection showed that all canals had been destroyed. There were twenty-six cases of pure lesions of the semicircular canals.

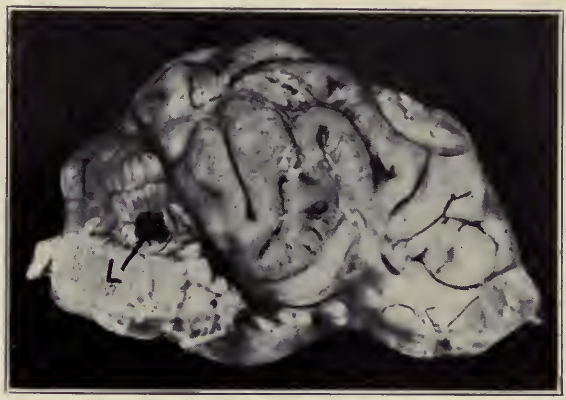

Fig. 11.—Type of lesion, *L*, produced by bone-wax tumors.

In nine cases one canal was destroyed six times, the lateral also called the horizontal, and three times the posterior vertical canal. In fifteen cases both of these canals were destroyed but numbers 48, 55 and 62 showed some degeneration due to opening the intracranial cavity.

In four cases all three canals were destroyed, but one of these showed an injury to the eighth nerve.

In nine cases, while trying to destroy all three canals, hemorrhage occurred from the bone, which was controlled by the use of bone wax.

An opening had been made just medial to the otic bone, and the bone wax projected through the dura and impinged on the cerebellum. These we have called cases of bone wax tumors (Fig. 11). Unfortunately, none of these brains was stained by Marchi, as they were thought not to bear on the problem, but on tabulation of our results it became evident that we had discarded a most valuable set of experiments. Consequently we deliberately made two such bone wax tumors, A 32 and A 33, to study the resulting degenerations.

In twenty-five cases the eighth nerve was cut. In fourteen cases, though an attempt was made to cut only the eighth nerve, more injury occurred, as was revealed by the subsequent histologic studies. In some of these cases Deiters' nucleus was injured, and in some there was a considerable lesion in the cerebellar nuclei; these injuries we believe account for the symptoms the animals presented. We feel justified in this conclusion as our pure eighth nerve lesions did not show these symptoms; Table 1, No. 48; Table 2, Nos. 89, 90, 92, 93, 94, 101, 108, 116, 130, 138, 135. Checking up the gross pathology by careful histologic studies we feel is essential in a research of this sort, and the results of previous observers are confusing and misleading because such studies were not made, at least not recorded in their papers.

In three cases we attempted to destroy Deiters' nucleus with a fine galvanocautery, but none of these lesions show pure Deiters' lesions.

In addition to the sixty-nine experiments on dogs, we have had for study one case in a man on whom we cut the eighth nerve for an intractable tinnitus and numerous clinical observations on the Bárány tests on intracranial cases.

Various methods have heretofore been used to cut the eighth nerve intracranially. The favorite method has been to go through the mastoid bone, pass a hook or knife in blindly and sweep it around. Biehl[13] and Thomas[12] are the only ones who have employed an intracranial approach by retracting the lateral lobe of the cerebellum and Van Gehuchten says he doubts if it can be done.

As one of us had repeatedly exposed the eighth nerve by this route in human beings and was opposed to operative procedures carried out in the dark, this method was used. The operative difficulties, however, are far greater than in human beings on account of the peculiar distribution of the blood vessels. This accounts for our high mortality in this group of animals. This operative approach necessitates retracting the cerebellum slightly, and even the gentlest retraction causes slight degenerative changes, as shown by the Marchi method. All operations were carried out under ether anesthesia.

TABLE 1.—RESULTS OF OPERATION

No. of Exp.	Operation	Ny-stag-mus	Devia-tion of Eye	Swing-ing of Head	Atti-tude	Fall-ing	Roll-ing	Ataxia	Circus Move-ments	No. of Days Lived	Stain	Microscopic Findings and Remarks
47	Exposure of otic bone; no canal injured	0	0	0	0	0	0	0	0	18	M	No degeneration
57	Exposure of otic bone; no canal injured	0	0	0	0	0	0	0	0	18	M	Not sectioned
	One Canal											
50	Right posterior vertical	2 →	0	0	0	0	0	0	0	18	M	No degeneration
56	Left horizontal	2 ←	0	0	0	0	0	0	0	18	M	No degeneration
61	Left horizontal	0	0	0	0	0	0	0	0	18	M	No degeneration (sections imperfectly stained)
124	Left horizontal	2 ↷	0	0	0	0	0	0	0	18	M	No degeneration (sections imperfectly stained)
126	Left posterior vertical	0	0	0	0	0	0	0	0	36	N	No chromatolytic changes
127	Left posterior vertical	2 ↑	4	0	0	0	0	0	0	35	N	No chromatolytic changes
132	Left horizontal	2 ←	4	0	0	0	0	0	0	18	M	No degeneration
A31	Left horizontal	2 ↷	4	0	0	0	0	0	0	18	M	Not sectioned
67	Right horizontal	2 →	21	0	0	0	0	0	0	21	M	No degeneration
	Two Canals *											
48	Right posterior vertical and right horizontal	2 ↷	0	0	0	0	0	0	0	18	M	Degeneration of a few fibers of eighth nerve. Skull opened in operation
51	Right posterior vertical and right horizontal	0	0	0	0	0	0	0	0	18	M	Specimen poorly stained
52	Right posterior vertical and right horizontal	1 ↶	0	0	0	0	0	0	0	18	M	No degeneration
55	Left posterior vertical and left horizontal	0	0	0	0	0	0	0	0	18	M	Flocculus destroyed. Fibers to cerebellar lateral nuclei
58	Left posterior vertical and left horizontal	0	0	0	0	0	0	0	0	18	M	Poorly stained
62	Left posterior vertical and left horizontal	2 ↷	18	0	2	0	0	5	0	18	M	Skull opened. Evidence of some intracranial injury. A few degenerated fibers in the pons (slight peduncular lesion)
63	Left posterior vertical and left horizontal	0	0	0	12	0	0	0	0	18	M	Specimen not properly penetrated
65	Left posterior vertical and left horizontal	0	0	0	0	0	0	0	0	18	M	Specimen poorly stained
72	Right posterior vertical and right horizontal	1 ↶	10	0	0	0	0	0	0	18	M	Poorly stained because of formaldehyd
73	Right posterior vertical and right horizontal	0	12	0	0	0	0	0	0	22	M	Not examined, as poorly preserved
76	Left posterior vertical and left horizontal	2 ↷	7	0	0	0	0	0	0	7	M and N	No degeneration and no chromatolytic changes
121	Left posterior vertical and left horizontal	2 ↷	28	0	0	0	0	0	0	28	M	No degeneration
133	Left posterior vertical and left horizontal	1 ↷	0	0	0	0	0	0	0	18	M	Specimen not examined
138	Left posterior vertical and left horizontal	2 ↷	0	0	0	0	0	0	0	18	N	No chromatolytic changes
131	Left posterior vertical and left horizontal	0	0	0	0	0	0	0	0	21	M	No degeneration
	Three Canals											
80	Left side	2 ←	Present	0	0	1	0	0	0	56	N	No chromatolytic changes
125	Left side	0	0	0	0	0	0	0	0	49	N	No chromatolytic changes
129	Left side	2 (?)	0	0	0	0	0	0	0	18	M	Slight injury of eighth nerve and degenerated fibers as seen in all cases of eighth nerve injuries
A29	Left side	2 ↷	1	0	0	0	0	0	0	17	..	Specimen lost

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* In each case the posterior vertical and horizontal canal was destroyed.

After the operation the dog was kept alive for eighteen days, and careful records kept of his symptoms. He was then anesthetized and killed by having his blood washed out with a solution of Muller's fluid containing 1 per cent. of formaldehyd. The brain and cord were then removed and suspended in Muller's solution, which was changed daily until it no longer became dark. In this way the brain was hardened rapidly and could be cut into thin slabs for immersion in Marchi solution (Muller's solution two parts, osmic acid 1 per cent. one part). We have used this method because we believe it shows nerve pathways with great accuracy, though it requires considerable experience to know how to interpret the findings. One of us (E. S.) obtained such satisfactory results by this method in previous studies on the nervous system that we have used it here. We believe it gives more accurate results than either the developmental method or the secondary atrophy method. The brains were then cut in serial celloidin sections and every third section mounted for study, the intervening sections being kept for reference if necessary. Two points are all important if this method is used: 1. There must be no formaldehyd in the specimen when placed in the Marchi solution, otherwise there will be a fine granular precipitate in the microscopic sections, which is very confusing. 2. Every degenerated nerve must be followed from one section to the other in order to avoid misinterpreting the findings.

A few specimens were hardened in alcohol and then stained with toluidin 1 per cent. for chromatolytic changes.

PURPOSE OF THE INVESTIGATION

In this research we have studied the following points:

1. Do fibers run directly from the ampulla into the medulla?
2. Symptoms produced by destruction of the semicircular canals.
3. The anatomic pathways of the vestibular and acoustic branches of the eighth nerve in the medulla, pons and cerebellum.
4. Symptoms resulting from cutting the eighth nerve.
5. Symptoms resulting from destruction of the nuclei of the eighth nerve.
6. Connections of the three semicircular canals with the eighth nerve nuclei in the medulla.
7. Pathways from the nuclei in which these nerves end to the cerebellum and cerebrum.

This paper does not deal with 5 and 6 as these points require a special series of experiments and will be made the subject of a subsequent paper.

TABLE 2.—SYMPTOMS FROM BONE-WAX TUMORS

No. of Exp.	Bone-Wax Tumors	Nystagmus	Deviation of Eye	Swinging of Head	Attitude	Falling	Rolling	Ataxia	Circus Movements	No. of Days Lived	Stain	Microscopic Findings and Remarks
53	Right	8 ↶	0	8	18	0	8	18	8	18	M	Poorly stained. Extensive lesion of cerebellum
59	Left	4 ↶	0	0	3	3	0	0	0	18	..	Not examined
64	Left	2 ↷	6	0	0	0	0	0	0	18	..	Not examined
66	Right	6 ↶	12	12	12	4	4	6	0	18	M	Series incomplete. Extensive lesion involving all of Deiters' nucleus
68	Right	0	21	0	21	0	0	0	0	21	..	Not examined
69	Right	4	12	0	0	0	0	0	11	25	..	Not examined
71	Right	0	14	0	0	8	0	0	0	22	..	Not examined
75	Left	3 ↷	3	2	3	0	0	2	0	7	..	Not examined
78	Left	3 ↷	3	18	0	0	0	2	0	33	..	Not examined
A32	Left	0	30	0	3	0	0	6	0	32	M	Extensive lesion of lateral lobes of cerebellum extending into the fourth nucleus. Deiters' nucleus and the inferior and middle peduncles have escaped. Very marked degeneration in fillet region
	Cautery to 4th Ventricle											
A1	......	0	0	0	0	2	0	2	0	5	..	Wound infected; lived six days. Too early for Marchi studies. Symptoms same as in those cases in which inferior peduncle is involved
A11	......	0	7	3	0	0	0	0	0	18	M	Lesion in posterior longitudinal bundles of both sides. Nucleus IX and inferior peduncle. Lesion too far caudal, too superficial to hit Deiters' nucleus
A21	......	0	4	0	10	0	0	0	0	18	M	Lesion in lateral cerebellar tract below region of Deiters' nucleus. Marked degeneration going up inferior peduncle

CONNECTIONS OF THE SEMICIRCULAR CANALS WITH THE MEDULLA

After some practice, one of us (B. Y. A.) perfected his technic so that it was possible for him to destroy either the posterior vertical canal or the lateral canal. It is not possible, however, to make an isolated lesion of the anterior vertical canal, and in order to study the result of the destruction of this canal all three canals were destroyed, and thus by a process of exclusion it was hoped to determine the course of the fibers supplying this canal. It was our hope that by studying the chromatolytic changes in the nuclei we might discover whether certain nuclei supplied certain canals. Our results along this line, however, were not convincing, and this phase of the problem still remains to be solved. We propose to attack the problem next by studying the secondary or indirect Wallerian degeneration and hope in this way to get some answer.

It has seemed probable to us that since there are three nuclei that compose the vestibular group, the nucleus of Deiters, nucleus of Bechterew and nucleus triangularis, each one of these is connected with one semicircular canal. If the various pathways that the Bárány followers use to explain the symptoms resulting from the stimulation of one canal are to be accepted, the connection of these nuclei with the cerebellum must first be worked out. Up to the present time there is insufficient anatomic basis for their deductions.

One of the disputed points about the canals has been whether after destruction of an ampulla degenerated fibers could be traced up the vestibular nerve. Tricomi-Allegra,[18] Matte [19] and Weigner [16] all claimed to have found such fibers, but a careful repetition of the work by Van Gehuchten [17] and his pupil Michotte do not bear this out. Our specimens bear out the observations of Van Gehuchten. In no case have we found degenerated fibers passing up the vestibular nerve after destruction of the semicircular canals.

We believe, therefore, that all fibers from the canals end in Scarpa's ganglion and that a second neuron carries impulses from here to the vestibular nuclei.

SYMPTOMS ARISING FROM DESTRUCTION OF THE SEMICIRCULAR CANALS

Practically all investigators have described a series of symptoms as characteristic of destruction of the semicircular canals. Wilson and Pike [19] enumerate them thus: "Deviation of one eye downward and outward, nystagmus with the slow phase to the injured side, inability to walk, falling to the injured side, head inclined to one side, falling to the injured side and going continually in a circle. At the end of the second week the animal stood erect with torsion of the head to one side and body inclined to one side."

TABLE 3.—RESULTS OF CUTTING EIGHTH NERVE

No. of Exp.	Operation	Nystagmus	Deviation of Eye	Swinging of Head	Attitude	Falling	Rolling	Ataxia	Circus Movements	No. of Days Lived	Stain	Microscopic Findings and Remarks
77	Right eighth nerve cut	4 ↶	18	7	7	4	0	7	0	18	M	Bilateral lesion of paraflocculus and flocculus. Small hemorrhage into upper portion of Deiters' nucleus. A few fibers to nucleus tecti, some fibers to posterior longitudinal bundle. Arcuate fibers to posterior corpora quadrigemina. Descending fibers to cord. Right eighth nerve cut
89	Right eighth nerve cut	0	0	0	0	0	0	0	0	9	..	No microscopic confirmation of operation. Too early for Marchi staining.
90	Right eighth nerve cut	0	0	0	0	0	0	0	0	18	M	Lesion mostly of vestibular portion. Slight lesion of flocculus. Fibers to nucleus tecti, few fibers to posterior longitudinal bundle. Arcuate fibers. No fibers to posterior corpora quadrigemina. Some fibers to cord. Right eighth nerve cut
92	Right eighth nerve cut	4 ↶	0	0	0	0	0	2	0	18	M	Small lesion of posterior dorsal and postventral paraflocculus and eighth nerve. A few fibers seen in posterior longitudinal bundle going as far forward as fourth nucleus; none to third. Fibers to Deiters', arcuate, posterior corpora quadrigemina and cord
93	Right eighth nerve cut	6 ↶	8	0	0	0	0	0	0	18	M	Small lesion of eighth and paraflocculus. Fibers to Deiters' to opposite corpora quadrigemina nucleus tecti, arcuate fibers and to cord. Fibers to posterior longitudinal bundle, but stop before they get near the fourth nucleus
94	Right eighth nerve cut	2 ↑	0	0	0	0	0	0	0	18	M	Specimen poorly stained. Eighth nerve cut but study of tracts not possible
100	Right eighth nerve cut	5 →	0	5	5	5	5	5	0	5	..	Injury to middle peduncle. Animal died on fifth day, too early for Marchi staining. These animals often die from exhaustion due to thrashing around. No other cause of death. Right eighth nerve cut
101	Right eighth nerve cut	3 →	0	0	0	0	0	0	0	3	..	Died too soon after operation for Marchi staining. Absence of symptoms indicates cerebellar nuclei and peduncles were not injured. Right eighth nerve cut
102	Right eighth nerve cut	3 ↶	3	0	0	0	3	0	0	3	..	Died too early for Marchi stain. Some evidence of injury to peduncle stained with thionin. Eighth nerve cut
108	Right eighth nerve cut	0	0	0	0	0	0	0	0	18	M	Eighth nerve not cut. Injury to flocculus and paraflocculus. Lesion did not extend into cerebellar nuclei. No degeneration except to cerebellar nuclei
116	Right eighth nerve cut	0	0	0	0	0	0	0	0	19	M	Injury to flocculus and paraflocculus lobules. Extensive degeneration to cerebellar nuclei, but no involvement of Deiters' or cerebellar nuclei. Some degenerated fibers in posterior longitudinal bundle in region of fourth nucleus. Eighth nerve cut
118	Left eighth nerve cut	3	3	3	3	3	3	3	3	3	..	Eighth nerve cut and inferior peduncle injured. Died too soon for Marchi staining. Died, apparently, from exhaustion as result of violent thrashing around
130	Left eighth nerve cut	3 ←	3	0	0	0	0	0	0	18	M	Partial lesion of eighth nerve, chiefly of cochlear portion. A few fibers to Deiters' nucleus; none to posterior longitudinal bundle; arcuate fibers and some to cord. A small bone-wax tumor present which produced a marked flocculus lesion

133	Right eighth nerve cut	1 →	1	0	0	0	0	1	0	1	..	Right eighth nerve cut. No evidence of other lesion in pons or Deiers' nucleus. Too early for Marchi staining. Death due to hemorrhage
138	On canals	1 ↷	0	0	10	0	0	2	0	14	M	This case originally belonged to Table 1, but the sections showed that in removing the entire otic bone the eighth nerve had been injured. Fibers to nucleus tecti, corpora quadrigemina of opposite side; peduncle and cerebellum uninjured. No fibers in posterior longitudinal bundle running up to sixth or fourth nuclei
A3	Right eighth nerve cut	8 ↶	10	2	10	0	0	0	0	18	M	Eighth and seventh nerves cut. Injury to flocculus and paraflocculus lobes with involvement of cerebellar nuclei; diffuse lesion not well localized; of value for the symptoms rather than the anatomic tracts
A5	Right eighth nerve cut	6 ↶	10	2	18	0	0	6	0	18	M	Lesion of Deiters' nucleus as well as eighth nerve. Fibers pass to anterolateral and contralateral portion of cord. Fibers to posterior longitudinal bundle, but none run forward to sixth and fourth nuclei. Fibers to corpora quadrigemina of opposite side and to cord
A8	Right eighth nerve cut	2 ↶	3	7	9	9	9	9	0	13	..	Wound infection. Eighth nerve cut. Rather extensive trauma to cerebellum. Too early for Marchi symptoms like those seen when peduncle and cerebellar nuclei are involved
A15	Right eighth nerve cut	5 ↶	5	5	5	5	0	0	3	5	..	Died of distemper; wound healed. Eighth nerve cut. Extensive trauma to cerebellum. Too early for Marchi staining. Symptoms like those seen in lesions of peduncles
A25	Right eighth nerve cut	2 ↶	2	0	0	0	0	2	2	2	..	Too early for Marchi staining. Eighth nerve cut. Large blood clot pressing on lateral lobe of cerebellum. Symptoms like those seen when cerebellar nuclei involved
135	Right eighth nerve cut	0	0	0	0	0	0	3	0	18	M	Eighth nerve cut, anterior and post paraflocculus slightly injured, no involvement of cerebellar nuclei. Fibers to Deiters' nucleus, nucleus tecti and corpora quadrigemina of opposite side. Fibers to posterior longitudinal bundle, but none to fourth and sixth nuclei. Some fibers to cord
136	Right eighth nerve cut	4 ↶	3	3	0	0	0	7	0	18	M	Eighth nerve cut. Hemorrhage into Deiters' nucleus. Poor Marchi penetration. Symptoms characteristic of lesion in region of Deiters' nucleus
A16	Right eighth nerve cut	5 ↶	16	4	6	0	0	10	0	18	M	Eighth nerve cut. Lesion of paraflocculus anterior and posterior quite extensive. Two microscopic hemorrhages in Deiters' region. Symptoms characteristic of Deiters' lesion. Fibers as in other cases of Deiters' lesions. Fibers to posterior longitudinal bundle; none go forward to fourth and sixth. Some fibers end in nucleus globosus and embolus in cerebellum
A18	Right eighth nerve cut	3 ↶	14	3	8	0	1	8	0	18	M	Infected hematoma. Eighth nerve cut. Small abscess extended into Deiters' region. Symptoms as in other Deiters' cases. Is confirmatory but in itself not a good case because of infection. Fibers as in other cases of Deiters' lesions. Fibers to posterior longitudinal bundle stop before they reach fourth and sixth nuclei
A26	Right eighth nerve cut	3 ↶	7	7	85	0	1	7	0	85	M	Eighth nerve cut. Lesion of Deiters' nucleus and inferior peduncle and anterior and posterior paraflocculus. Fibers in posterior longitudinal bundle and corpora quadrigemina and nuclei tecti. A few fibers traced to region of descending root of fifth nucleus around aqueduct of Sylvius
A33	Left eighth nerve cut	3	10	0	3	0	0	3	0	31	M	Pure lesion of eighth nerve. Fibers go to Deiters' nucleus and stop there; a few arcuate fibers. Nothing found histologically to explain ataxia or attitude of head

We have observed these symptoms in the eight cases of bone-wax tumor and also in cases in which we attempted to cut the eighth nerve intracranially, but in every case in which other symptoms than nystagmus or deviation of the eye occurred there was an injury either to the nuclei of the cerebellum or peduncles of the pons. In fact, it is possible by looking over Table 1 to pick out from the symptoms those cases in which there were other lesions.

These symptoms, as we have observed them, may be grouped under eight headings:

1. *Nystagmus.*—In every case in our series in which this phenomenon has been observed it has been bilateral, of the so-called labyrinthine type, that is, consisting of a slow movement of the eyeball in one direction and a quick movement in the opposite direction. In some instances the motion was purely lateral or vertical and in others it was definitely rotatory in character; this we speak of as clockwise and anticlockwise motion.

We have seen a definite relation between the side of the lesion and the direction of the nystagmus. In all left-sided lesions the slow component was toward the left side where purely horizontal motion was observed; and the slow phase was in the clockwise direction, that is, the top of the eye moved toward the animal's left when rotatory nystagmus occurred. With right sided lesions, the opposite was observed.

Nystagmus was the most frequently observed symptom. It was usually seen as soon as the animal recovered from the anesthetic, when it was in some cases very rapid, and in most cases it had entirely disappeared within seventy-two hours, often within twelve hours, though it sometimes persisted for four or five days.

2. *Deviation of Eye on Side Corresponding to Operation.*—The deviation was usually downward and outward. In rare instances downward and inward. Occasionally the eye on the opposite side showed some deviation downward, but in all animals of this series this symptom has been rarely observed.

The degree of deviation is markedly influenced by the position of the animal's head, frequently being unnoticed when the animal is at rest in the attitude of choice. It is usually increased by pointing the snout upward or by turning the head in such a way that the operated side is uppermost. This is the most persistent symptom observed in the entire study, usually remaining unchanged throughout the eighteen days during which the animal was kept alive after operation.

3. *Swinging of Head.*—This striking symptom was observed only when the animal was held up by grasping it about the thorax and holding it in such a manner that the snout pointed almost vertically. The animal would then swing its head about in a circle, first bending its neck

toward the operated side, then backward, then toward the opposite side, then forward and then to the starting point. This movement in well marked cases was carried out with great rapidity and violence and kept up till either the animal or the observer was exhausted. The tendency to swing the head thus was soon lost and usually was not observed for more than three or four days.

It was not observed in any animal with a simple labyrinthine lesion.

4. *Attitude of Head.*—The head is rotated about its long axis in such a way that the injured ear points to the corresponding shoulder. In some more marked cases the neck is flexed toward this side, sometimes to 90 degrees or more. This peculiar attitude did not always become apparent in the first twenty-four to forty-eight hours and usually persisted till the animal's death, though it gradually diminished and possibly would have entirely disappeared had the animals been kept alive for a much longer time.

5. *Falling toward the Operated Side.*—This symptom apparently was due to a weakness in the legs on the side corresponding to the lesion and to spasticity of the opposite extremities. When this symptom occurred it usually persisted only for from twenty-four to seventy-two hours after operation.

6. *Rolling about Long Axis of Body.*—The direction of the rolling is dependent on the side of operation. Starting from the normal resting position the animal rolls over and over toward the operated side, finally coming to rest with the operated side downward.

7. *Ataxia.*—This was observed when the animal was walking about.

8. *Circus Movements.*—Animals exhibiting this symptom had a tendency to go round in circles as about a ring and were unable to proceed by a direct course from one point to another. The accompanying tables show the number of animals of this series exhibiting the various symptoms described.

In pure lesions of the semicircular canals, however, the only symptoms we observed were nystagmus and occasionally deviation of the eye on the operated side. The type of nystagmus was not always the same when a certain canal was destroyed; thus, in Experiments 124 and A31 there was clockwise nystagmus when the left horizontal canal was destroyed, while in Experiments 56 and 132 it was lateral. The same variation in symptoms was observed when two canals were destroyed, but it was striking that whereas nystagmus occurred in all cases in which one canal was destroyed, it often was absent when two canals were destroyed. For this observation we have no explanation to offer, but merely record the fact. The same symptoms were observed when all three canals were destroyed. In no case did the nystagmus last more than two days.

From these experiments we feel justified in concluding that in all previous experiments the varied symptoms ascribed to destruction of the canals must have been due to intracranial injuries which could only have been discovered on microscopic studies. Our contention is supported by the experiments on the eighth nerve for in those cases also no such symptoms as are described by Wilson and Pike[19] and others were observed unless there were other injuries of the nervous system. A small intracranial hematoma in some of our cases was enough to produce these symptoms.

Baginsky,[8] thirty-five years ago, in referring to von Bechterew's work, pointed out that the symptoms were due to an injury of other regions.

ANATOMIC CONNECTIONS OF VESTIBULAR AND ACOUSTIC BRANCHES OF THE EIGHTH NERVE IN THE MEDULLA, PONS AND CEREBELLUM

Whereas the cochlear branch of the eighth nerve has frequently been studied, the vestibular portion has been sadly neglected. In going over the literature, we have been impressed with the varied descriptions of the nuclei of the vestibular nerve. Our experiences bear out, however, the statements of other observers that the fibers pass to the three nuclei, Deiters' (Fig. 1), Bechterew's and nucleus triangularis. In a personal communication with Dr. Elliot Smith of University College, London, he told us that he has been teaching his students that on account of the confusion existing in regard to the vestibular nuclei he has applied the term Deiters' nucleus to the entire group of cells. This we are not willing to do as yet as there are such varied types of ganglion cells in these nuclei that we hope to be able to show that they have different functions.

Our specimens show fibers passing from the vestibular nerve to the nucleus tecti, (Fig. 8) as claimed by Cajal.[24] Our specimens also show degenerated fibers which pass from the paraflocculus lobe (Elliot Smith[25]) to the nuclei of the cerebellum (Fig. 4). The degeneration appears in all of our specimens and is due to the slight pressure on the paraflocculus while retracting it to expose the eighth nerve. As we have seen no fibers passing beyond the vestibular nuclei, we believe that redistribution of the fibers takes place after they end in these nuclei. This brings us to the neurons passing from the vestibular nuclei upward.

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24. Cajal: Beitr. zum Studium der medulla oblongata, Leipzig, 1896, p. 75-101. Die Endigungen des Aüserem Lemniscus, oder die sekundäre akustische Nervenbahn, Deutsch. med. Wchnschr. **28**:275, 1902. Les ganglions termineaux du nerf acoustique des oiseaux, Jour. Psychol. u. Neurol. **13**:214-239, 1908.

25. Smith, G. E.: The Primary Subdivision of the Mammalian Cerebellum, J. Anat. & Physiol. **36**:375. Notes on the Morphology of the Cerebellum, J. Anat. & Physiol. **17**:329, 1902-1903.

In our series we have six lesions of Deiters'[26] nucleus (confirmed microscopically)—Experiments 77, 136, A5, A16, A18 and A26. These have varied in size; thus A16 had only two-minute hemorrhages. In addition the animals in Experiments 53, 66, 75, 78, 100, 102, 118, A3, A8, A15 and A25 had the same symptoms that the patients with confirmed Deiters' nucleus lesions had. In some of these cases, as shown in the tables, histologic studies were made and showed extensive lesions of the cerebellar nuclei as in Experiments A3, A8, A15, and it is fair to suppose that all these animals had a lesion either in Deiters' nucleus, in the cerebellar nuclei, or in the pathways connecting the two. The symptoms did not enable us to differentiate lesions in these various regions. It should be mentioned that the late Victor Horsley and R. H. Clarke made some elaborate studies about fourteen years ago on the anatomy and physiology of the cerebellar nuclei, but unfortunately these were never published. Their studies contained evidence which might have enabled one to differentiate lesions in these different regions.

A21 and A1 are of great interest, however, in that in these animals the lesion lay in the pons but caudal to Deiters' nucleus, and in these cases the symptoms were few. This indicates that destruction of the fibers running cephalad from Deiters' neucleus are responsible for those symptoms about which there has been so much discussion. This observation is in accord with those of Fraser.[15] In none of our lesions were all the cells of the acoustic portion of the eighth nerve destroyed. In all cases we found fibers passing into the cord, as described by Fraser and Lloyd[27] and Russell[28] and Mott.[29] These fibers pass to the anterolateral portion of the cord on the same side, and a few cross over to the anterolateral region of the contralateral side. A few fibers were seen passing to the posterior longitudinal bundle of the same and opposite sides (Fig. 7). These ended just before reaching the region of the fourth nerve, and none was seen to go further forward (Fig. 8). We have been unable to confirm the observations of Fraser that large numbers of fibers pass up the posterior longitudinal bundle. In none of our cases, however, were the lesions as large or extensive as those that Fraser produced, which are illustrated in his paper.

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[26] The nucleus which bears Deiters' name was so named by his teacher, Max Schultze, who edited Deiters' work, as Deiter died very suddenly as a young man before completing his work. According to the illustration which Schultze published, and which we reproduce, it seems to us that the portion of the nucleus is that to which Bechterew later attached his own name.

[27] Lloyd, R. E.: On Chromatolysis in Deiters' Nucleus After Hemisection of the Cord, J. Physiol. **25**:191, 1900.

[28] Russell, Risien: The Origin and Destination of Certain Afferent and Efferent Tracts in the Medulla Oblongata, Brain **20**:409, 1897.

[29] Mott, F. W.: Experimental Enquiry upon the Afferent Tracts of the Central Nervous System of the Monkey, Brain **18**:1, 1895.

It is of great importance to note that these fibers lie close to the floor of the fourth ventricle and continue to lie so superficially as they go cephalad that any pressure, such as from an internal hydrocephalus, may readily affect them. We have had several patients with hydrocephalus without other lesions, as shown by necropsy, in whom the Bárány tests showed abnormalities. We believe that the internal hydrocephalus associated with almost all posterior fossa lesions is responsible for the abnormalities observed in the Bárány tests.

A large group of fibers are seen passing up to the cerebellum and seem to end in the nucleus tecti (Fig. 8). We have not seen any fibers pass to the lateral lobes of the cerebellum either of the same or opposite side. Our results do not show any connection with the lateral lobes of the cerebellum. Dr. Elliot Smith confirms this observation, and he has informed us that observations by Gordon Holmes (as yet unpublished) on a large number of gunshot wounds of the cerebellum, have shown no demonstrable disturbances of the arms, legs, or trunk as Bárány asserts occur when there are lesions of certain portions of the lateral lobes.

We have found arcuate fibers running across the median line (Fig. 7) ending in the superior olive, as most other observers have found. A few pass on into the lateral fillet and end in the inferior portion of the posterior corpus quadrigeminum of the opposite side (Fig. 9). Van Gehuchten asserted that these fibers end before they reach the corpus quadrigeminum, but we have traced them directly to that region. We have found no fibers that run more anteriorly and have found no fibers that go to the external geniculate body.

SYMPTOMS RESULTING FROM CUTTING THE EIGHTH NERVE

In the twenty-seven experiments in which we cut the eighth nerve a large number have had to be considered under a different heading since more was destroyed than merely the eighth nerve. These cases can be identified almost as accurately by the symptoms as by a study of the anatomic specimens. The cases that appear at the end of Table 3, in which the animals showed swinging of the head, circus movements, etc., all had lesions of other regions in addition to destruction of the eighth nerve.

The only symptoms presented by patients with pure eighth nerve lesions were deviation of the eye downward and outward, nystagmus with the slow phase anticlockwise and in three cases, Experiments 92, 135 and A26, a transient ataxia in walking which was so slight that we had some doubt as to its existence. We thought at first the inevitable slight injury to the paraflocculus might be responsible for this ataxia, but a more careful analysis of our cases and the total absence of this symptom in certain cases of injury to the paraflocculus definitely contradicts that idea.

One clinical case supports these experimental findings. On this patient the eighth nerve was cut for distressing tinnitus. His symptoms were lateral nystagmus to either side and slight ataxia for a few days. These symptoms are similar to our experimental results. Certainly, cutting the eighth nerve in man produces none of the symptoms that have been reported by others when the eighth nerve is cut in animals.

In seventeen experiments the lesion was in the region of Deiters' nucleus. These animals all showed:

1. Deviation of the right eye and facial paralysis.
2. Rotatory nystagmus counterclockwise.
3. Swinging of the head.
4. Inability to stand.
5. Marked ataxia.

Some animals showed the symptoms for longer periods than others. When the injury was not so severe there was a certain amount of readjustment apparently, while in others there was no evidence of this. We consider it important to disregard symptoms that are present only a few hours after operation. There must be a certain degree of permanency in order to consider them of value. Some observers have described symptoms that were noted when the animals came out of the anesthesia; this we have not done.

The symptoms we have noted are those described by von Bechterew and others as characteristic of an eighth nerve lesion, but we believe we have shown that the symptoms of rotation often called circus movements are not produced by cutting the eighth nerve but are due to injury to neighboring structures, especially the middle and superior peduncles of the cerebellum. The so-called cerebellar attitude of the head seen in these animals is due to injury of the middle peduncle and not, we believe, a protective mechanism as has been claimed by Cushing. That the cerebellar attitude is due to involvement of the peduncle was shown by Victor Horsley more than ten years ago.

In all our lesions of the eighth nerve we have noted deviation of the eye down and out. We are unable to find a satisfactory explanation for this.

The great difference between our results and those of others we believe to be due to the fact that they operated in such a manner as to be unable to see exactly what they did, and as careful histologic studies were not made, at least are not recorded in any of these articles, the injuries responsible for these symptoms were overlooked.

SYMPTOMS ARISING FROM BONE-WAX TUMORS

For the sake of completeness we add a few remarks about this group of cases. The symptoms which these animals presented are for the most part the same as those presented by our animals with Deiters' nucleus lesions. Unfortunately, as stated previously, most of these cases were not studied histologically but in Expriment A32 such studies were made. The sections of this animal are very instructive for, while the lesion of the cerebellum is extensive and extended into the cerebellar nuclei, the inferior peduncle and middle peduncle escaped injury. The severe symptoms of rolling and circus movements seem to be directly dependent on injury of the inferior peduncle. This was the case in Experiments 77, 100, 118, A8, A15 and A26.

CONCLUSIONS

1. There are no fibers running directly from the semicircular canals to the nuclei of the vestibular nerve.

2. The fibers running from Deiters' nucleus anteriorly in the posterior longitudinal bundle are few in number and stop before they get to the third and fourth nuclei so that it is doubtful whether there is any connection between Deiters' nucleus and the other nuclei. This is the oculovestibular tract spoken of by Wilson and Pike.

3. We have found no fibers passing from Deiters' nucleus to the lateral lobes of the cerebellum where the centers Bárány has described lie.

4. All anatomic evidence to support Bárány's contention as to the connections between Deiters' nucleus and the cerebellum is lacking.

5. There are fibers from Deiters' nucleus that end in the posterior corpus quadrigeminum of the opposite side, but no neuron goes directly to the external geniculate.

6. All fibers of the vestibular nerve end in one of the three vestibular nuclei or in the nucleus tecti of the vermis.

7. Circus movements, rolling over and over, ataxia, swinging of the head, attitude of the head, hitherto described as due to a lesion of the eighth nerve or destruction of the semicircular canals, are due to injuries to the cerebellar nuclei or middle peduncle.

8. The so-called cerebellar attitude of the head is due to involvement of the middle peduncle.

9. Nystagmus and deviation of the eye downward and outward are the only constant symptoms observed after pure lesions of either the semicircular canals or eighth nerve.

10. As the vestibular fibers lie so near the floor of the fourth ventricle, the symptoms described by Bárány as due to isolated lesions in the pons may be produced by, and probably usually are due to, the internal hydrocephalus which is a common accompaniment of posterior fossa lesions.[30]

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[30] In addition to the footnotes given, the following may be of interest:

Brown, A. Crum: On Sense of Rotation and Anatomy and Physiology of Semicircular Canals, J. Anat. & Physiol. **8**:1875.

Bruce: Tracts of Mid and Hind Brain, Edinburgh, 1892.

Ewald, R.: Zur Physiol. der Bogengänge, Pflügers Arch. **41**:1888.

Flechsig: Zur Lehre vom zentralen Verlauf des Hörnerven, Neurol. Centralbl., 1886. Weitere Mitteilung über die Beziehung des Unteren Vierhugels zu dem Hörnerven, Neurol. Centralbl., 1890.

Forel: Zur Acusticus Frage, Neurol. Centralbl., 1887.

Hoyt, Randal: Studies on the Morphology of the Gray Matter, Neurol. Bull. **1**:227, 1918.

Leidler, R.: Untersuch. über das Endungsgebiet des N. Vestib., Arb. a. d. neurol. Inst. a. d. Wien Univ. **19**:262, 1912-1913.

Loeb, Jacques: Ueber den Antheil des Hörnerven an den nach Gehirnverletzung auftretenden Zwangsbewegungen, Arch. f. d. ges. Physiol. **50**:66, 1891.

Malesani: Sulle degenerazioni dei centri nervosi dei Colombi all estirpazione dei canali semicircolari, Le nevraxe **10**:341, 1908.

Malone: Ueber die Kerne des Menschlichen Diencephalon, Neurol. Centralbl. **29**:290-300, 1910.

Martin: Zur Endigung des Nerv. acust. im Gehirn der Katze, Anat. Anzeig. **9**:1893.

Marx. Herman: Untersuch über Kleinhirn Veränderungen nach Zerstörung der häutigen Bogengänge des Ohrlab, Pflügers Arch. **120**:166, 1907.

Roller: Die Zerebral Verbind d. dritten bis zwölften Hirnnerv, Ztschr. f. Psychol. **35**: 1904. Eine Aufsteigende Acusticuswurzel, Arch. f. mikr. Anat. **18**:403, 1880.

Sabin, Florence R.: On the Anatomical Relations of the Nuclei of Reception of the Cochlear and Vestibular Nerves, Johns Hopkins Hosp. Bull. 1897, p. 253.

Sala: Sull 'origini del nervo acustico. Sep. Abdr. aus Monitoro Zoologico Italiano, 1891.

Sewell, H.: Experiments on Ears of Fishes, with Reference to Function of Equilibrium, J. Physiol. **4**:1883.

Tozier and Sherrington: Receptors and Afferents of the Third, Fourth and Sixth Cranial Nerves, Proc. Royal Soc. **82**:450, 1910.

A STUDY OF THE CORTICAL OLFACTORY CENTER

BASED ON TWO CASES OF UNILATERAL INVOLVEMENT OF THE OLFACTORY LOBE

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It is many years since Broca and Zuckerkandl first claimed that the gyrus hippocampi, cornu ammonis, and gyrus fornicatus were the cortical olfactory centers. Experimental study, clinicopathologic observations, and comparative anatomy have been productive of a large number of papers concerning this important subject. In spite of the efforts of many authors, the exact location of the cortical olfactory center cannot be said to have been definitely determined. Recently I had an opportunity to study two patients presenting unilateral involvement of the olfactory lobe, one of congenital absence, the other of softening due to arteriosclerosis. Both had a lesion in the same area of the homolateral lobus pyriformis, the lesion being reasonably regarded as a secondary degeneration following the primary involvement of the peripheral olfactory center.

REPORT OF CASES

Case 1.—*Clinical History.*—Case No. 22048, Danvers State Hospital; necropsy No. 2165. A man, born in August, 1861, single, whose early life and development were normal, and who had received a common school, possibly a high school, education; who had worked as a letter collector; when about 25 years of age had had some form of convulsions, followed by several others in the course of a few years. Two years previous to admission he had the first attack in a number of years. While working in May, 1919, he fell and was carried home unconscious. On Jan. 2, 1920, he had another attack and was taken to the hospital. Becoming somewhat restless and disturbed, especially at night, he was transferred to the Danvers State Hospital.

Examination.—He was fairly well developed but poorly nourished. His chest was somewhat paralytic; respiratory movements were regular. Throughout both sides of the chest were a few scattered râles. Heart sounds were weak and regular. The pulse rate was 75 per minute; good quality. The blood pressure was systolic 140, diastolic 90. Nothing unusual was noted on palpation of the abdomen. The urine was normal; the Wassermann reaction was negative.

At times he complained of attacks of vertigo. The conjunctiva was somewhat infused; the pupils were irregular and reacted promptly to light and accommodation. The visual field was somewhat restricted. On account of the poor cooperation of the patient, the sense of taste and of smell could not be examined. The knee jerks were somewhat increased. Superficial reflexes were apparently retained. The facial muscles showed some lack of motility. There was considerable loss of power in both hands. The Romberg sign was positive. There was gross tremor on exertion.

Course of Disease.—In the ward he was talkative, elated, restless, disoriented and more or less confused. This condition persisted for two or three days when he became dull, depressed, stupid and apathetic. He gradually began to lose interest in things about him, and it was with great difficulty that he could be made to take sufficient nourishment. He remained practically in the same condition until Jan. 22, when he died, the cause of death being bronchopneumonia.

Postmortem Examination.—Necropsy was performed eight hours postmortem. Anatomic diagnoses were: emaciation, cyanosis of fingers, phimosis, chronic adhesive pleuritis, bronchopneumonia, hypertrophy of left ventricle, sclerosis of aorta, chronic passive congestion of liver, chronic diffuse nephritis, hypertrophy of prostate, chronic pachymeningitis and leptomeningitis, slight atrophy of central and frontal convolutions and absence of the right olfactory lobe.

Head and Brain.—The scalp was thin, and adherent in the frontal region. The calvarium was thick and dense, diploë being absent. The internal table was eroded along the longitudinal sinus. The groovings for the middle meningeal arteries were rather faintly marked. The dura mater showed marked thickening, and was slightly adherent to the calvarium. The pia mater was thickened and clouded over the vertex, especially in the frontal and central regions. Moderate subpial edema was noted in these regions. The convolutions of both hemispheres were slightly atrophic, the sulci being somewhat widened. The carotids, basilar and major arteries of the cerebrum and cerebellum were only slightly thickened. No atheromatous patches were noted.

The right olfactory bulb with its peduncle was missing. At the root of the olfactory sulcus a small elevation about the size of the head of a pin was found, corresponding to the olfactory trigon. On the right side the stria olfactoria, medialis and lateralis could not be identified, while on the left were well marked whitish striations. Substantia perforata anterior of the right side was markedly deeper than that of the left. Lobus pyriformis, gyrus hippocampi, cornu ammonis, and gyrus fornicatus were alike on both sides and did not show any malformations or agenesis. A difference of the convolutional pattern was noted in the frontal poles. Sulcus frontomarginalis of Wernicke was prominent on the right, while on the left it was rudimentary. Otherwise there was no gross difference in the hemispheres, which were equal in weight (150 gm. after fixation). Frontal section, made after Meynert, revealed nothing remarkable in any of the planes. The fossae of the cribriform plates, where the olfactory lobes rested, showed an abnormal condition corresponding to the unilateral absence of the olfactory bulb. The left side was wide and flat, as in a normal person; the right side was very shallow with little space for the olfactory bulb.

Nothing unusual was noted in the pituitary body, the pons, cerebellum and medulla.

The spinal cord showed nothing of note.

Microscopic Examination.—Practically all parts of the lobus pyriformis, gyrus hippocampi, and cornu ammonis were submitted to special investigation. Several blocks from the gyrus cinguli and isthmus of the gyrus fornicatus were carefully studied, as well as other important areas of the cerebral cortex. Thionin staining, Weigert-Pal's method for staining myelin sheaths, Mallory's method for neuroglia demonstration, Herxheimer's scharlach r staining, and several other general staining methods were employed.

Outside the rhinencephalon, the microscopic examination showed only minor alterations. The pia mater over the vertex was thickened, but there was no perivascular cellular exudate. In the frontal area, precentral and postcentral convolutions, was a small number of more or less degenerated ganglion cells, the stratigraphical picture not being much altered. Neuroglia cells and fibers were noticeably increased generally. Marginal neuroglia fiber nets were thickened. A well marked envelop formation was observed around the large cortical vessels. The myelin sheaths showed no marked involvement. In the entire limbic lobe of the right side, the most marked alteration was found in the lobus pyriformis, lateral to the gyrus circumambiens and anterior to the uncus proper.

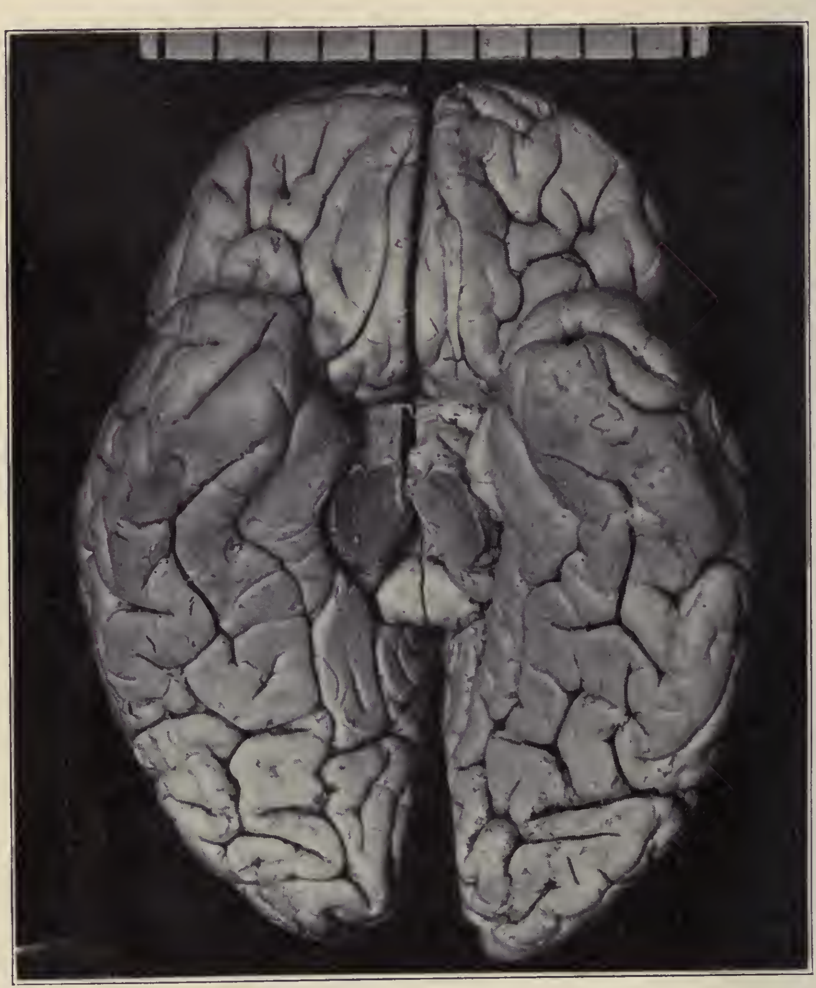

Fig. 1.—Brain without the right olfactory lobe.

Before taking up the pathologic findings, I shall give in brief the normal histology.[1] As is known to histopathologists, the lobus pyriformis presents a large number of peculiar cell nests in the small pyramidal cell layer, giving a bizarre appearance to the cortex. The cell nests consist of islets of small pyramidal cells and islets of giant polymorphonuclear cells, the latter being more prominent. Cajal[2] attributes an important olfactory function to the area over which the peculiar cell islets are distributed. In place of medium and large sized pyramidal cells there are pyramidal cells of equal size with more or less deviated apical prolongations. Below this is a narrow layer standing out on account of the sparsity of nerve cells. Still deeper we come to the large and small fusiform layer, as in the other cortex. The zonal layer is well developed and composed of dense fibers. Corresponding to the islets of cells described, circular areas sparsely supplied with fibers are found. The line of Baillarger is wide but its fibers are sparse.

The clusters of large polymorphous cells had for the most part disappeared, showing only a few scattered cells which were no longer in the formation of nests. The cytoplasm, as well as the nucleus, was much shrunken. The body plasma stained uniformly dark and Nissl bodies were barely visible. The nuclei were irregular in outline and located in the periphery of the cell body, either toward the apex or the side of the cell. The clusters of small pyramidal cells were equally involved, and the cells were difficult to identify. In consequence of the above condition the lamina, containing the characteristic cell clusters, often tended to show blank areas almost bare of nerve cells. The number of cells in the following layer were noticeably decreased; this frequently showed areas devoid of ganglion cells. Many cells were chromophilous and shrunken, their dendrites being traced for a considerable distance; a small number, however, were stained pale and presented a shadow-like appearance (Zellschattenfigur). The large and small fusiform cells of the deeper layer were uniformly shrunken with a few advanced forms of degeneration.

The tangential fibers in the plexiform layer were greatly involved, containing many degenerated fibers and a few swollen elements. Corresponding to the layers of cell clusters and underlying cells, the myelin sheaths showed the most marked degeneration. Radial, as well as inter-radial fibers had undergone degeneration, leaving pale stained areas. The fibers in the line of Baillarger were barely identified. The medulla was somewhat paler than normal with apparently degenerated fibers.

An extreme fibrillar gliosis was found in the plexiform layer, in the lamina of cell clusters, and in the upper part of the pyramidal cell layer. Fine fibers were densely interwoven throughout these layers, showing no definite demarcation between these and the neuroglia network of the cell-free border. In the deeper cell layers and in the upper part of the white matter were numerous spider-cells with luxuriant fibrillation, the fibrils being of rather small caliber.

The whole picture suggested a chronic long-standing process, apparently the result of the nonfunctioning of this part of the brain. It must be emphasized that no marked alterations of any kind were found in the same area of the contralateral hemisphere of the brain.

In the cornu ammonis, when compared with the healthy side, slight cell sparsity was found in the stratum cellularum pyramidalium and the terminal plate. The cells showed well marked fatty pigmentous deposits, mostly in the apical prolongations. However, such deposits were found in cells of the healthy side. Myelin sheaths showed no marked degeneration. The neuroglia elements were not remarkable. No decided alterations were demonstrated in the rest of the limbic lobe.

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[1] Campbell: Histologic Studies on Localization of Cerebral Function, New York, G. P. Putnam's Sons, 1905.

[2] Cajal: Histologie du systeme nerveux de l'homme et des vertèbres. Ed. 2, Paris, 1911.

COMMENT ON CASE 1

In the literature, cases showing the absence of the olfactory bulb are rare. Cases were reported by Bernard,[3] 1858; Kundrat,[4] 1882; Duval,[5] 1884; Valenti,[6] 1911; Ranke,[7] 1913; Weidenreich,[8] 1914; Haga,[9] 1915, and Tanaka,[10] 1917. With the exception of Tanaka's patient, no one of these was subjected to a research investigation of the supposed center of the olfactory sense. Tanaka found most marked alterations in the cornu ammonis and less marked, but fairly well-pronounced, changes in the hippocampal gyrus proper. He avoided the conclusion, however, that the changes found were due to the secondary degeneration following the primary arrest of development of the olfactory lobes—since his patient was an epileptic, and the same changes would be found in epileptic brains without involvement of the olfactory apparatus. It is interesting to know that the present patient also showed epileptic convulsions in earlier life. (This will be referred to later in the comment on Case 2).

Weidenreich's patient, although deprived of both olfactory lobes, was said to have possessed some olfactory sense during life. The olfactory nerves must have taken some abnormal course into the cortical center. It is to be regretted that in the present patient the olfactory sense was not tested, as in Tanaka's case. The question may arise, particularly in this case, as to whether or not the cortical olfactory center developed normally, also whether the nerve tract ran an abnormal course to the peripheral olfactory apparatus. To this I can give no definite answer, but the long-standing degenerative process, found in the supposed cortical olfactory area, would suggest the nonfunctioning of the unilateral olfactory tract.

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[3] Bernard, Claude: Leçons sur la physiologie et la pathologie du système nerveux **2**:1858.

[4] Kundrat: Arhinencephalie, 1882; abstr. in Schwalbe: Die Missbildungen des Kopfes, 1913.

[5] Duval: Bull. Soc. d'Anthropol. de Paris, Ed. 3 **8**:1884.

[6] Valenti: Un cas d'absence unilaterale de l'appareil olfactif, Arch. ital. de biol. **64**:457, 1911.

[7] Ranke: Zytoarchitektonik der Grosshirnrinde in einem Fall von Kyklopie, 1913; Referat in Folia neurobiologica, 1913.

[8] Weidenreich: Ueber partiellen Riechlappendefekt und Eunuchidismus beim Menschen, Ztschr. f. Morphol. u. Anthropol., 1914.

[9] Haga: Eenzijdig ontbreken van den tractus olfactorius, Nederl. Tijdschr. v. Geneesk. Jaargang 1915, No. 22, pp. 2450-2452.

[10] Tanaka: Absence of Lobus Olfactorius and Sclerosis of Cornu Ammonis, Arch. Neurol. & Phychiat. **3**:151 (Aug.) 1920.

The cause of the defect of the olfactory lobe is not known. Ernst [11] holds the compression theory, while Schwalbe [12] attributes this condition to embryonal defect. Weidenreich interpreted his case as resulting from the failure to combine the olfactory nerve and the olfactory lobe, in consequence of which the latter tends to atrophy. In the present case the cribriform plate of the right side showed some deformity. This condition would probably suggest the difficulty which had existed in effecting the combination of central and peripheral olfactory organs, which, according to His, occurs in a relatively later embryonal stage.

CASE 2.—*Clinical History.*—Case No. 22265, Danvers State Hospital; necropsy No. 2202. A man, born in Kittery, Me., March, 1863, married and the father of six children, had a negative family history for mental disease. Little was known about his early life and development. He was a general laborer, used alcohol to some extent, and was a heavy smoker. The onset of the present psychosis began following a "shock," when he became stupid and dull. From that time he had had periods of marked confusion, occurring about once a month and simulating epileptiform seizures. More recently he suffered a more severe attack, when he remained in bed four days, and showed a marked loss of memory. It was impossible to hold his attention for any length of time on any subject, and he showed a tendency to irrelevance and desultoriness in conversation. While at home he frequently expressed a desire to go home. It was thought he had auditory and visual hallucinations. He complained that some one was trying to get him into trouble. On admission to this hospital he was quiet and affable, but showed a marked deterioration, especially in the memory field. He was slightly confused and markedly disoriented. Attention was extremely difficult to gain and hold; he showed considerable paresis of thought with a tendency to repetition of questions, and words, with more or less incoherence. For the greater part of the time he remained quietly in bed, giving little trouble except for occasional untidy habits. He had no insight into his condition; judgment was markedly impaired, but no definite delusions were elicited. He had some suspicious ideas, but these were not clearly expressed or fully formulated.

Examination.—He was fairly well developed and moderately nourished. On the left side was an inguinal hernia. Supraclavicular and infraclavicular fossae were prominent on both sides. Heart: the apex was palpable in the fifth left interspace. There were no abnormal heart sounds. The peripheral arteries showed marked sclerosis. The blood pressure was: systolic, 210; diastolic, 98. The abdominal organs were normal. Urine: it contained albumin and a few hyaline casts. The Wassermann reaction in the blood serum was negative. There was slight ptosis; arcus senilis was present. The pupils were regular and reacted to light and accommodation. He could not hear a watch tick when held close to his ear. Sense of taste and of smell were normal. There were no disturbances of motility and sensibility. The knee jerks were slightly diminished. He showed a slight speech defect and inequality of the pupils but no definite paralytic symptoms in the arms or legs. He frequently stated that his head seemed twitchy. As far as surroundings were concerned he was disoriented, and only approximately correct in the matter of time.

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[11] Ernst: Missbildungen des Nervensystems, in Schwalbe: Morphologie der Missbildungen des Menschen und der Tiere, Lief., Part 3, No. 2, 1910.

[12] Schwalbe und Josephy: Die Missbildungen des Kopfes, in Schwalbe: Morphologie der Missbildungen des Menschen und der Tiere, Part 3, No. 2, 1913.

Course of Disease.—July 9, 1920: The patient had a typical grand mal convulsion while in the dining room. Sept. 22, 1920: During September the patient had several convulsions, followed by much confusion. Nov. 3, 1920: The patient died at 3 a. m., the cause of death being given as chronic diffuse nephritis and arteriosclerosis.

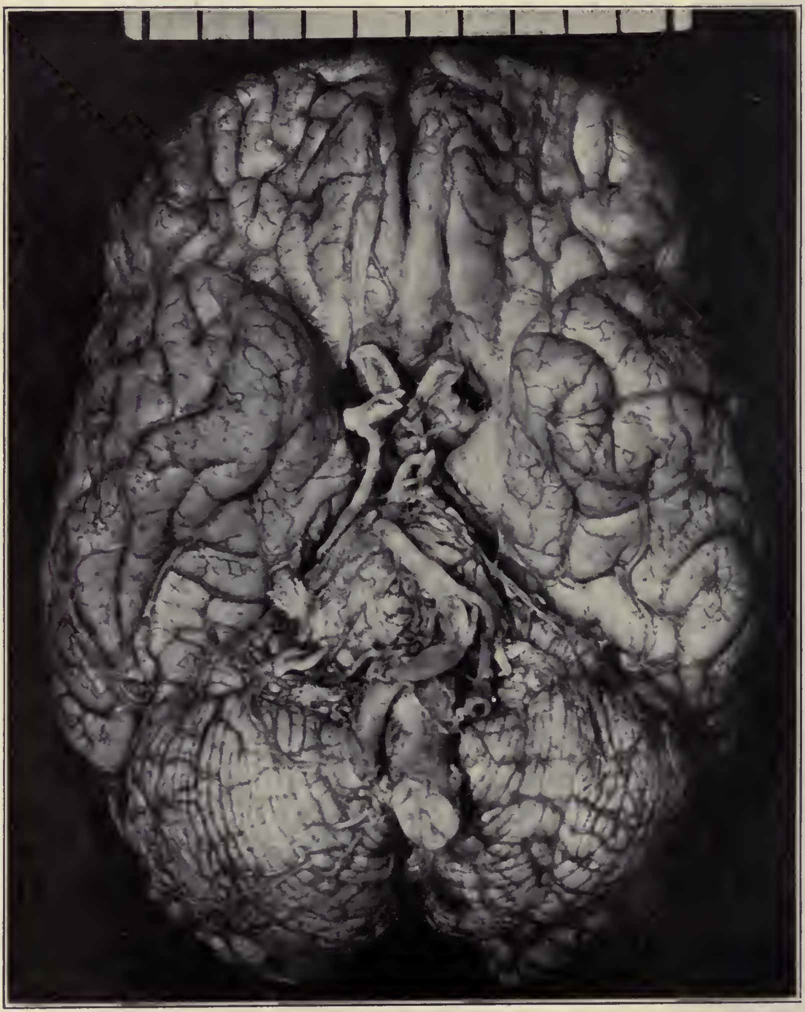

Fig. 2.—Softening of the left olfactory lobe.

Postmortem Examination.—Necropsy was performed five hours after death. Important anatomic diagnoses were: Decubitus in trochanter and sacral regions, chronic adhesive pleuritis, old tuberculous process of the right apex, early bronchopneumonia, sclerosis of aorta and coronary arteries, chronic diffuse nephritis, fatty change of liver, chronic pachymeningitis and leptomeningitis, sclerosis of cerebral arteries, left olfactory bulb missing and softening of the left olfactory tract.

Head and Brain: The calvarium was dense; diploë was absent. The dura mater was thickened and adherent to the calvarium throughout. Pachyonian granulations were prominent. The brain weighed 1,410 gm. The pia mater was slightly thickened and opaque along the course of the large vessels. The vertebral arteries were markedly sclerotic on both sides, and noticeably dilated at a point 2 cm. below the junction. The basilar artery presented considerable sclerosis with whitish-yellow patches, which were for the most part calcified. The carotids and major arteries of the cerebrum and cerebellum were all thickened, atheromatous and calcified patches being strewn throughout. The right posterior cerebral artery was given off from the carotid, and a posterior communicating artery was found between the posterior cerebral artery and the terminal branching of the basilar artery.

The left olfactory bulb presented well marked degeneration. The bulb was replaced by a thin grayish thread, embedded in a thickened pia mater. The left olfactory tract was thinner than the right, grayish-yellow and extremely soft in consistency. The lateral olfactory striations could be traced for a distance across the substantia perforata anterior, toward the lobus pyriformis.

The convolutions did not appear to be atrophic, except in some small regionary areas. The lower surface of the middle frontal convolution on the left side was atrophic and brownish, suggesting a softening. On the lateral surface of the left occipital lobe was also found a small softening. The convolutional pattern was not much different on either side. With the exception of the anomaly of the circle of Willis, described in the foregoing, there were no noticeable malformations in the central nervous system. The lobus pyriformis, gyrus hippocampi, cornu ammonis, and gyrus fornicatus presented nothing unusual. The pons, cerebellum, medulla and spinal cord presented nothing remarkable.

Microscopic Examination.—The limbic lobe and the other important cortical areas were studied by the same methods as in Case 1. The arteries of the pia mater and cortex showed sclerotic changes. Proliferation of adventitia, proliferative as well as regressive alterations of the endothelium, occasional calcification of vessel walls and arteriofibrosis were encountered throughout. Occasionally small cortical vessels presented the appearance of hyaline degeneration. Irregular thickening of the marginal neuroglia network, a tendency toward wedgeshaped marginal gliosis, perivascular, cellular and fibrillar gliosis were also common findings. Focal areas of softening were observed in the left frontal pole and left occipital lobe. Perivascular thinning of the myelin sheaths was noted throughout, particularly in the left frontal lobe. In the neighborhood of the sclerotic vessels the nerve cells displayed various stages of regressive changes. A majority of the cells were shrunken with eccentric, irregular, chromophilous nuclei. Fatty pigmented degeneration was found in conjunction with the sclerotic cell changes. In short, changes encountered in general indicated an arteriosclerotic nature of the alteration.

Section of the olfactory tract of the left side revealed a well marked degeneration of the myelin sheaths, as well as of the axis cylinders. Few fibers remained uninvolved at the level of the olfactory trigon.

In the lobus pyriformis of the left side a remarkable alteration was encountered. The plexiform layer had become considerably narrower. The cells of the clusters were noticeably decreased in number, some clusters having apparently disappeared; some cells were shrunken with dark eccentric nuclei, while others were slightly swollen with chromatolysis and occasional vacuole formation. The equal sized pyramidal cells were greatly reduced in number, those remaining presenting various grades of degeneration; also slightly swollen cell bodies with chromatolysis and eccentric nuclei were encountered. The cells of the fusiform layer were fairly well preserved, although a number of cells showed marked regressive changes.

The tangential fibers were greatly involved; those remaining ran irregularly, and some were markedly thickened. In the layer of cell clusters and the lamina of pyramidal cells Weigert preparation showed marked reduction of radial, as well as of inter-radial, fibers. Quite large sized blank areas, without demonstrable fibers, also came to view. The nerve fibers in the medulla were apparently fewer than in the corresponding part of the other hemisphere.

The neuroglia fibers were increased, not only in the cell-free border, but also in the deeper cortical layers. The fibers were thicker than those observed in Case 1, and the network less dense. A considerable number of satellite cells were encountered, especially around the nerve cells of the deeper layers. Some cells were accompanied by more than nine satellites; two or three satellites were occasionally seen along the apical prolongations. The glial reaction, therefore, indicated a relatively fresh and still progressive degenerating process.

The blood vessels were more or less sclerotic, showing both regressive and progressive changes. However, the cell and fiber degeneration did not seem to be associated with the vascular alteration. The parenchymatous devastation was diffuse throughout the anterior part of the lobus pyriformis. The alteration of this particular part, described in the foregoing, should be considered as a secondary degeneration following the unilateral softening of the olfactory bulb and tract.

The cornu ammonis and the rest of the limbic lobe showed a slight degeneration, both sides being equal in intensity. The cell and fiber degeneration was usually found around, or in the neighborhood of, the sclerotic vessels. Most of the cells showed cell sclerosis combined with fatty pigmented degeneration, while no swollen cells with vacuoles or chromatolysis were encountered. There was slight fibrillar and cellular increase of neuroglia, especially along the cortical margin and around the sclerotic vessels. These changes were, in all probability, due to the arteriosclerotic condition of the vessels. It was almost impossible, however, to determine whether or not the olfactory degeneration displayed any secondary changes in this part of the cortex.

COMMENT ON CASE 2

Undoubtedly Case 2 was one of arteriosclerotic brain disease. The lesion of the left olfactory lobe should, by all means, be considered as softening due to arteriosclerosis. The whole picture of the lesion found in the homolateral lobus pyriformis suggested secondary degeneration following the primary involvement of the olfactory lobe. The history of the case revealed nothing abnormal concerning the olfactory sense of the patient. It is doubtful, however, whether the sense of smell was tested unilaterally.

The patient in Case 2 had typical epileptiform convulsions. The patient in Case 1 and Tanaka's patient were epileptic subjects. It is a question whether or not the secondary involvement of the cortical olfactory center might cause the epileptiform seizures. Both Tanaka's case and Case 1 were of a congenital nature and, therefore, the seizures might be attributed to it, as defective subjects are liable to show epileptic convulsions. The epileptic seizures of the patient in Case 2 might also be attributed to the arteriosclerotic condition, as this is often the case. To answer this question a study of cases of uncomplicated olfactory degeneration is desirable. For this purpose I searched 2,230 necropsy reports of the Danvers State Hospital Laboratory material, and found a case showing bilateral olfactory degeneration (of unknown nature) without arteriosclerosis or recognizable malformations. This patient (No. 18596, necropsy No. 1824) showed an absence of the olfactory sense, and the most typical acquired form of epileptic convulsions. The brain, however, was in such a condition that careful study of the finer structure was impossible, and nothing definite could be ascertained. However, I am inclined to believe in olfactory center degeneration as the possible cause of epilepsy, rather than as a coincidental complication.

SUMMARY

Although certain parts of Broca's limbic lobe are known to govern the sense of smell, opinions as to the exact area of the cortical olfactory center are manifold.

Gorschkow [13] demonstrated, by experiment, that the ablation of the lobus pyriformis would be attended by loss of the sense of smell on the same side and diminution on the opposite. In a case of tumor at the base of the right temporal lobe, destroying the uncus and gyrus hippocampi, reported by Siebert,[14] substances used to test the olfactory sense were falsely interpreted. In Jackson and Beevor's [15] case of tumor at the tip of the temporal lobe, involving the anterior end of the lobus pyriformis and the nucleus amygdalae, the patient was said to have been constantly afflicted with a sense of horrid smell. Cajal, from histologic study of human and vertebrate brains, reached the conclusion that the important olfactory center lies in the regio olfactiva of the lobus pyriformis, and that the cornu ammonis, focus spheno-occipitalis, the subiculum and the focus praesubicularis represent probably the tertiary olfactory center. Bechterew regarded the lobus pyriformis, by experimental study on dogs, as the perception center, and the neighboring regions, such as the subiculum and cornu ammonis, as the conception center. Campbell concluded that the lobus pyriformis must almost certainly be regarded as the chief olfactory center.

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[13] Gorschkow: Uber Geschmacks- und Geruchscentren in der Hirnrinde, Inaug. Dissert., St. Petersburg, 1901; abstr. Neurol. Centralbl. 1901.

[14] Siebert: Ein Fall von Hirntumor mit Geruchstäuschungen, Monatschr. f. Psychiat. u. Neurol. **6**:1899.

[15] Jackson and Beevor: Case of Tumor of the Right Temporo-Sphenoidal Lobe Bearing on the Localization of the Sense of Smell and on the Interpretation of a Particular Variety of Epilepsy, Brain **12**:346, 1890.

Hill,[16] on the other hand, pointed out that in some anosmatic animals the lobus hippocampi attains a moderate degree of development, and therefore he believed that this lobe cannot exist for the control of this function solely. He attached far more importance to the fascia dentata of the cornu ammonis, because this is the only structure in this region which is wholly wanting in anosmatic animals. Edinger,[17] also from the comparative anatomic standpoint, seems to believe that the cornu ammonis is the chief cortical olfactory center.

In both cases of unilateral olfactory lobe involvement, herein presented, I found a well marked degeneration of the anterior part of the lobus pyriformis. The first case was, however, that of an epileptic subject, and therefore the changes might be of an epileptic nature and not of a secondary degeneration. The second case was undoubtedly that of an arteriosclerotic brain disease, but the changes in the lobus pyriformis were different than in other parts, indicating their secondary nature. Considering these two cases, it is not unreasonable to conclude that the degeneration of the lobus pyriformis in the first case was also secondary. From the study of these two cases, and also from data presented by various observers, I am inclined to believe that the lobus pyriformis must be the chief olfactory center in the human brain.

The cornu ammonis and the rest of the limbic lobe showed little evidence of degeneration, and I have nothing to offer, from the present study, as to the functional importance of these parts.

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[16] Hill: The Hippocampus, Philadelphia Trans. **184**:1893.

[17] Edinger: Vorlesungen uber den Bau der nervosen Zentralcrgane, 1911.

A CASE OF FRIEDREICH'S ATAXIA

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AND
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Friedreich's ataxia is still a rarity, and the opportunity to examine the spinal cord in an early stage of the disease does not often present itself. The following case is of interest because it occurred in a boy, aged 11 years, who died of an acute intercurrent disease. He had a younger brother who also had this familial affection. The fact that they were negroes perhaps lends additional interest, for Friedreich's ataxia is probably even more rare in the negro than in the white race.

The problem which the disease presents is not so much the distribution of the lesions as the causation and exact nature of the affection. As is well known, the disease process is not confined to the posterior columns; it is more diffused, and is evidently a degeneration with neurogliar proliferation, but the causation is obscure and has given rise to a variety of theories, none of which is particularly convincing. The fact that it occurs in several children of the same family, suggests, of course, some obscure familial defect; but the additional fact that numerous brothers and sisters often escape, indicates that the cause, whatever it is, is strangely limited or selective in its action. Gowers' theory that the disease is due to an "abiotrophy," or developmental defect, has been received with some favor, although it is not so much an explanation as a mere statement of a fact, for the problem remains as to what is meant by an "abiotrophy," and why it acts, as in the present instance, in only two out of five children; and in still another instance, which one of us has recently seen, in two out of eight children. That the disease is not due to an infection seems certain. Syphilis can be ruled out.

There are some physiologic problems also, as, for instance, the cause of the ataxia, the possible rôle of the cerebellum, and the absence in many cases of pronounced involvement of sensation. Possibly the degeneration so frequently found in the cells of Clark's column and in the direct cerebellar tract may explain the peculiar character of the ataxia. That it is not due to cerebellar disease is indicated by the fact that the cerebellum has not been found diseased in most of the cases examined. The absence of tactile and other modes of anesthesia has been ascribed by A. W. Campbell[1] to the escape of the posterior roots and the sensory tracts in the cord. The spinothalamic tract is not

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1. Campbell, A. W.: M. J. Australia **1**:135 (Feb. 17) 1917.

involved. The degeneration sometimes seen in the posterior roots and even in the cells of the posterior ganglions may be merely a consequence of the posterior column lesions. A possible resemblance of the lesions to those of multiple sclerosis has been suggested. The two lesions have, however, little in common. The lesions of multiple sclerosis are primarily focal in character and irregular in distribution. Friedreich's ataxia is a system disease, but the widespread glial activity shown in the present case at least suggests that there is also here, in some sense at least, a primary gliosis.

The location of the lesions in the posterior columns in the present case suggests a resemblance to what is seen in locomotor ataxia, but that the disease is not tabes is generally recognized. We do not mean to stress this resemblance here, but we venture to call attention to what seems to be an involvement especially of the third series of fibers as observed in the process of myelinization. This resemblance is shown on comparing the photographs with those of Trepinski, a student of Flechsug.[2] Whether it has any significance, we do not know.

The involvement of one of these systems, usually the third, to the exclusion of the remainder of the posterior column fibers, is common in tabes, but probably is not infrequent in other forms of posterior column gliosis. In this case it may mean nothing more than a fiber destruction corresponding to the posterior root involvement. Which is the earlier of the two is a subject for speculation. It is interesting to note that the microscopic structure of the replacement glia is here totally different from that occurring in tabes.

Much has been written about the possible relationship of Friedreich's ataxia to Marie's so-called cerebellar ataxia. Such a relationship is not indicated in the present case, for neither the cerebellum nor the inferior cerebellar peduncle is involved. If there is any relationship between the two diseases it would seem to depend on some common obscure developmental defect acting on different structures in different cases.

It has been suggested[3] that the location of the lesions in the posterolateral regions of the cord may be due to a less adequate blood supply, the supposition being that therefore the resistive power is less. This idea was suggested by Williamson, who pointed out that these regions receive their blood supply from small meningeal branches, instead of from a deep central artery as in the anterior and central parts of the cord. But this is not an adequate anatomic reason for supposing differences in the extent of vascularization of the parts of the cord, unless such a conclusion is to be drawn from the situation of the cord lesions in pernicious anemia, as Campbell has suggested. The lesions in Friedreich's ataxia are, however, quite unlike those in pernicious anemia.

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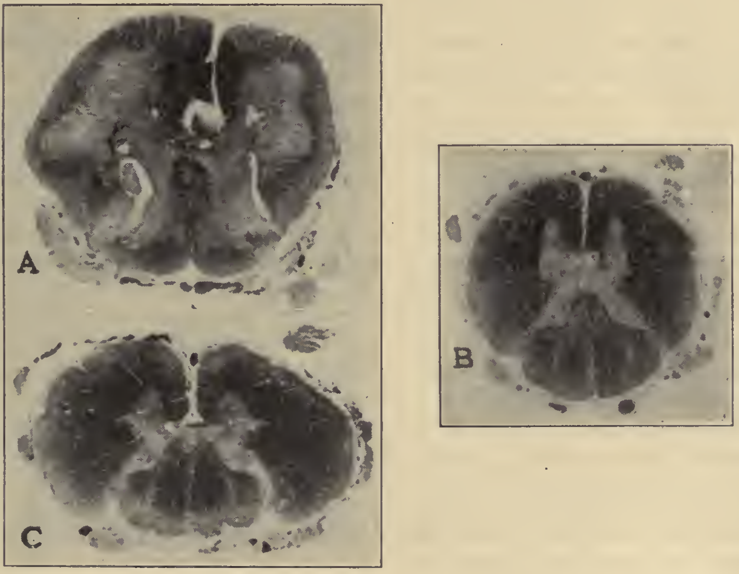
[2] Barker: The Nervous System, New York, G. E. Stechert & Co., p. 430.

[3] Williamson: Diseases of the Spinal Cord, New York, Oxford University Press.

CLINICAL REPORT BY DR. LLOYD [4]

Family History.—The two brothers, one of whose cases is here reported, were full-blooded negroes. They were well developed and intelligent for their age. The father, aged 39, and the mother, aged 35, were in good health. The blood of both the mother and an older daughter gave a negative reaction to the Wassermann test. The blood of the father could not be secured, but the test of the mother's blood was more important for determining the question of congenital syphilis. The blood and spinal fluid of both the patients were negative. The mother had had five children, a daughter, aged 14, living and well; two sons, the patients, and another son who had had convulsions and died at 3 years of age; and a daughter, aged 3, who was living and well. There had been one miscarriage. Otherwise the family history was unimportant. There had been no ataxia in collateral branches, so far as could be ascertained.



Weigert sections of different levels of the cord, showing involvement of the third myelogenetic system in the posterior columns. The peripheral paleness is an artefact.

Case 1.—J. C., the elder brother, aged 11 years, was the one whose brain and cord were examined. He was born in normal labor, and walked and talked at the usual age. He had convulsions in infancy and the usual diseases of childhood. He went to school for a while and did well. The ataxia began when the boy was 8. He became awkward in the movements of his legs, falling on his knees when attempting to walk. Scoliosis was present. When he was admitted to the hospital, in the autumn of 1919, his gait was very ataxic, he kept his feet far apart, swayed violently, and required assistance in walking. It was not the gait of locomotor ataxia. He had the Romberg sign, and well marked adiadokokinesis. The finger-to-nose and heel-to-knee tests showed ataxia. The knee and Achilles' reflexes were absent, and there was no Babinski reflex. The abdominal and cremasteric reflexes were preserved. Superficial sensation in all its modes was normal, that is, to touch, pain, heat and cold. There was apparent loss of ability to recognize a tuning fork held on the radius, ulna, or tibia. There was no shortening of the plantar arch, no hammer toe, as reported in some cases; no muscular atrophy, no pain, no girdle sense, no loss of spincter control. Speech was hesitating and staccato rather than drawling. Vision was normal, the pupils equal, the reaction to light sluggish, the action on accommodation and convergence prompt. Dr. Shannon reported a curious lack of pigmentation in the upper nasal quadrant of the iris of the right eye, also in the upper nasal and lower temporal quadrants of the left iris, suggesting iritic atrophy. On distant fixation the left eye rotated outward, although on close fixation fair convergence was obtained, indicating a condition of exophoria. This measured about 12 degrees with the Maddox rod. Ataxic nystagmus was not elicited. In the right eye the media were clear, the disk discolored and somewhat atrophic, the margins clearly outlined, the vessels moderately contracted, the macula normal; there were no changes in the periphery of the fundus. The left eye was in a similar condition. A few months after his admission to the hospital this boy died of influenzal pneumonia.

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[4] From the Neurological Wards of the Philadelphia General Hospital. The patients were shown at a meeting of the Philadelphia Neurological Society, Nov. 21, 1919. See report of transactions, Arch. Neurol. & Psychiat., March, 1920.

Case 2.—The younger brother, aged 9, was born in normal labor, walked and talked at the usual age, went to school for a while and was considered bright. He had convulsions at the age of 2 years and the usual diseases of childhood. The ataxia began when he was 6 years old and when he was recovering from an attack of typhoid fever. His gait and most other symptoms were similar to those of his brother. He had the ataxic gait, with swaying movements and unsteady station, which, however, was not increased on closing his eyes. The deep reflexes were abolished. There was no pain and no loss of superficial sensation in any of its modes; there was slight loss to the tuning fork. Unlike his brother, he did not have scoliosis. There was no muscular atrophy, no loss of sphincter control. Speech was slightly hesitating. The eyes were in a similar condition to that of his brother. Nystagmus was seen when the eyes were fixed on a moving object, especially in extremes of rotation. The light reflex was sluggish, accommodation normal.

COMMENT

It is noteworthy that speech defects and nystagmus were not marked in these boys, but these symptoms are sometimes rather late in appearing in Friedreich's ataxia. The slight appearance of optic atrophy is also noteworthy. Several cases have been reported in which the light reflex was lost, and Gowers thought that this was an evidence of syphilis. It might occur in juvenile tabes, but the present cases were evidently not instances of that disease.

The occurrence of convulsions in three children in this family is not to be overlooked. The two boys and another brother had been thus affected. It may at least indicate an unstable nervous system.

HISTOLOGIC REPORT BY DR. NEWCOMER [5]

The gross appearance of the brain and cord was not remarkable. Sections were cut from the various cord levels, from the cerebellar cortex, including the anterior and posterior superior worm, from the cerebellar nuclei and from six areas of the cerebral cortex. In sections from the cortex stained with toluidin blue, there were some distinct vessel fibrosis and perivascular infiltration, partly with leukocytes and partly with round cells which were perhaps glial in origin. There was a slight general gliosis, some areas contained nerve cells with a number of satellite cells, rarely more than six or seven, but enough to attract attention. The nerve cells and sections from the basal ganglions showed nothing remarkable, except that not infrequently they were almost filled with pigment. The sections from the cerebellum were normal. In the superior worm, the Purkinje cells were normal and as numerous as usual. In the medulla, the nerve cells contained considerable pigment, less in the olive. Sections from the cord showed marked pigmentation of the anterior horn cells. The lateral horn cells showed some simple chromatolysis. There were no normal Clark's column cells in numerous sections from the dorsal cord. They were either absent, or, when present, were much diminished in number and markedly sclerosed; many gave evidence of considerable chromatolysis. There was considerable gliosis of the column. In the cord there was the same vessel fibrosis, but not the infiltration seen in the cortex.

Sections stained with phosphotungstic acid hematoxylin, with the exception of the cord, showed nothing further that was remarkable. Sections of the cord gave evidence of a considerable gliosis of the posterior columns which involved, in all the levels, substantially the region of the third myelogenic system as described by Trepinski,[2] and in which the nerve fibers were practically all gone. This distribution is seen in the photographs of the Weigert specimens. These illustrations show the posterior column degeneration only. In the rest of the posterior columns there was a gliosis of less intensity. The gliosis in the region of the greatest scarring consisted of a dense fibrous mat with rather small inactive glia cell nuclei—a mat of whorls and irregularly arranged glia fibers. There were some spider cells, but none of the columnar fiber arrangement so typical of tabes. In the less involved regions of the posterior columns the gliosis was also largely of fibrous character, but spider cells were more numerous. In all the levels of the cord there was some gliosis of the crossed pyramidal tract. This gliosis was fibrous in character but contained a number of rather large spider cells. Nerve fiber replacement was appreciable in the center of the tract. At the border of the cord in the region of the dorsal cerebellar tract there was a certain amount of spider cell gliosis, and the glia fiber strands were heavier and more conspicuous than anywhere else along the border of the cord. In the region of the direct pyramidal tract there were some gliosis and large spider cells. This slight gliosis of the direct pyramidal tract decreased as we progressed toward the dorsal and lumbar region. There was more gliosis than usual in the region of the anterior commissure. In the gray matter of the cord there was strikingly more than the usual number of glia cells—many of them were large spider cells quite active in appearance. The gliosis in the gray matter was greatest, both fibrous and cellular, in the lower region of the cord, dorsal and lumbar. It was less in the posterior horns than elsewhere. There was considerable fiber loss with extensive spindle cell replacement in the posterior nerve roots and considerable glia fiber invasion of the proximal portion of the nerve roots. The fiber loss was greatest in the cervical region, involving most of the fibers and decreased gradually to become least in the sacral region where only a few fibers were gone. The anterior roots were intact.

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[5] From the Laboratory of the Pennsylvania Hospital, Department for Mental and Nervous Diseases, Philadelphia.

With the acid fuchsin—light green stain, the extensive cord gliosis was distinct, and it was possible to estimate the fiber loss in the direct cerebellar tracts as being about one sixth. The crossed pyramidal tracts were about the same. The posterior column gliosis was, if anything else, less than this except in the region of the third system in which the fiber loss was practically complete. In the ventral field more than half the fibers were gone.

There was thus a general vessel fibrosis and some perivascular infiltration, of which at least the latter may be considered as due to the terminal disease (influenzal pneumonia). There was a general marked nerve cell pigmentation and a gliosis of the gray matter of the cortex and cord, and in addition there was a tract gliosis involving almost completely the fibers in the posterior columns having the position of the third myelogenic system and slightly the direct cerebellar and crossed pyramidal tracts. There was extensive posterior nerve root degeneration. The involvement of the direct cerebellar tract was too slight to show in the Weigert preparations of the restiform body, and there was nothing abnormal in the appearance of the dorsal portion of the superior worm, nor was there any gliosis of this or other portions of the cerebellar cortex.

COMPARATIVE RESULTS OF COLLOIDAL MASTIC AND COLLOIDAL GOLD TESTS *

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Although the colloidal mastic test, first introduced by Emmanuel,[1] and later modified by Cutting,[2] has been in limited use for several years, the question of its value as compared with that of the colloidal gold test is still an unsettled one. Considering the more complicated and uncertain methods of preparing the reagents used in the colloidal gold test, the simplicity and uniformity of the mastic reaction has much to recommend it. If it can be shown that the information provided by the mastic test is equivalent to that given by the gold test, or that the mastic test may in some cases provide information which the gold test fails to give, the test deserves wider use than is now made of it. In either case, the mastic test should prove of value not only when facilities for performing the gold test are lacking, but also as a check on the more complicated gold reaction in the laboratories where this test is now employed.

Both the gold and mastic reactions apparently depend on colloidal phenomena. The gold solution undergoes quantitative colorimetric changes leading up to complete precipitation, while the suspension of finely divided colloidal mastic particles undergoes quantitative flocculation, also culminating in complete precipitation. Resins other than mastic may be employed for the preparation of the colloidal suspension, the successful substitution depending on a proper adjustment of other reagents employed in the test, to balance variations in the amount of free acids present in other resins. It seems probable that these free acids exercise a dominating influence on the reaction.

Recent authors have thoroughly reviewed the literature, making a further complete survey unnecessary. Stanton [3] and Camp [4] conclude that the results obtained with the mastic test are in close agreement with those obtained with the gold test.

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* From the Syphilis Department of the Medical Clinic, Johns Hopkins Hospital.

1. Emmanuel, G.: Eine neue Reaktion zur Untersuchungen des Liquor cerebrospinalis, Berl, klin. Wchnschr. **52**:792, 1915.

2. Cutting, J. A.: A New Mastic Test for the Spinal Fluid, J. A. M. A. **68**:1810 (June 16) 1917.

3. Stanton, J. M.: Concerning the Colloidal Mastic Test, Arch. Neurol. & Psychiat. **4**:301 (Sept.) 1920.

4. Camp, C.: Colloidal Mastic Test on Cerebrospinal Fluid, Am. J. Syph. **4**:301, 1920.

In order to secure further information regarding this still unsettled point, we have studied the spinal fluids of 311 patients. Gold[5] and mastic tests were compared with each other and with the cell counts, globulin reactions and Wassermann tests in all the fluids. The material is almost entirely from the syphilis department, and consists of fluids from patients exhibiting various forms of syphilis. A small number of fluids from nonsyphilitic patients, on whom puncture was performed to rule out neurosyphilis, has also been examined. Of the 311 fluids, there was complete agreement between the gold and mastic tests 234 times. In the remaining seventy-seven instances in which there was disagreement, the discrepancies have been analyzed.

TECHNIC

The technic which we have employed in the mastic test is that described by Stanton.[3] It is essentially as follows: Ten grams of commercial gum mastic are dissolved in 100 c.c. of absolute alcohol, and the resultant cloudy fluid filtered several times until a clear straw colored solution is obtained. This stock solution is kept in glass stoppered bottles at room temperature. The emulsion is prepared with 1 c.c. of the stock solution added to 9 c.c. of absolute alcohol, which is then added, with gentle mixing, to 40 c.c. of once distilled water.

In setting up the test ten small tubes are employed. To the first tube in the series are added 1.5 c.c. of a stock salt solution (99 c.c. of 1.25 per cent. sodium chlorid solution plus 1 c.c. of 0.5 per cent. potassium carbonate solution), and to each of the remaining tubes 1 c.c. is added. In the first tube there is placed 0.5 c.c. of the spinal fluid to be tested. After mixing this dilution of spinal fluid, a titration is made throughout the series by transferring 1 c.c. from the first to the second tube, from the second to the third, etc., 1 c.c. being finally discarded from the last tube. Finally, 1 c.c. of the mastic emulsion is added to each tube. After mixing, the tubes are allowed to stand over night at room temperature, and the results are read in the morning. No special precautions regarding absolute cleanliness of glassware, such as are necessary for the gold test, need be observed.

In recording results, previous experimenters have used either the terms positive and negative, or, as later suggested by Smith,[6] a numerical scale denoting degrees of flocculation and precipitation, as follows: No change is indicated by 0; loss of opalescence and very slight precipitation by 1; milky fluid with distinct precipitation by 2; marked precipitation with turbid fluid by 3; and complete precipitation by 4.

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[5] The colloidal gold tests were all performed in the laboratory of the Phipps Psychiatric Clinic of this hospital, under the supervision of Dr. Phyllis Greenacre, for whose cooperation we are deeply grateful. The technic employed is that described by L. D. Felton in "A Study of the Specificity of the Colloidal Gold Reaction from the Physicochemical Standpoint," Trans. Sect. Path. and Physiol., A. M. A., 1917, p. 73.

[6] Smith, E. R.: The Mastic Reaction on the Cerebrospinal Fluid, Med. Rec. **92**:675, 1917.

With this scale it is possible to chart curves which correspond in type to those of the gold test, but as they differ numerically, 4 denoting in the mastic test complete precipitation, which in the gold test is recorded as 5, we deemed it expedient as a preliminary to our work to construct a scale which would exactly correspond to that employed everywhere in the gold test. By observing the reactions obtained in a number of fluids in parallel series of gold and mastic tests the following scale was arrived at: 0, opalescence, no change; 1, milky fluid with no precipitation; 2, milky fluid with slight precipitation; 3, milky fluid with moderate precipitation; 4, cloudy fluid with almost complete precipitation; and 5, clear fluid with complete precipitation. With this scale mastic reactions recorded as 2 may occur in normal fluids with about the same frequency as in the gold test, and such reactions are therefore regarded as within normal limits. Fluids which produce moderate precipitation, however, as denoted by a reading 3, we have tentatively regarded as abnormal for reasons which will be given in the analysis of this type of reaction. Slight color changes occurring in normal fluids, as observed in the gold test, occur more frequently with some preparations of colloidal gold chlorid solutions than with others, but a color change reading as high as 3 is regarded as abnormal and is recorded as a syphilitic zone curve.

We have observed three types of curves with the mastic test: a paretic curve, a negative curve and a "mastic 3" curve. In only one instance was a curve of the gold syphilitic zone type observed. For the purpose of comparison with the gold reaction, we have arranged our results in seven groups as follows:

RESULTS OF AUTHORS' TESTS

Gold and Mastic Curves Both Negative.—In this group are 203 of the fluids examined. In general, there was complete agreement with the clinical findings and with the other tests of the cerebrospinal fluid. In only one instance was there a positive Wassermann reaction in the fluid with both colloidal tests negative; this was a fluid with 24 cells and a positive globulin reaction in a patient with secondary syphilis with irregular pupils and sluggish reflexes. Cell counts were not made in all fluids, but in thirteen instances there was an increased cell count or an increased globulin content or both. The diagnoses of the patients in this group were: primary syphilis, 11; secondary syphilis, 45; tertiary syphilis, 42; syphilis Wassermann, 34; latent syphilis, 7; congenital syphilis, 6; neurosyphilis (mostly treated cases), 21; undiagnosed (except in one instance all referred from other clinics for spinal puncture), 14; and nonsyphilitic patients, 23. The nonsyphilitic patients composed a diagnostic group including normal persons investigated for familial syphilis, neurasthenia, lethargic encephalitis, transverse myelitis and other neurologic affections. Practically all the syphilitic patients had received some treatment before the time of puncture, which probably accounts for some of the negative curves in those fluids showing other minor abnormalities.

TABLE 1.—Gold and Mastic Curves Both Paretic *

Case No.	Diagnosis on Admission. Form of Syphilis	Blood Wassermann Reaction	Cerebrospinal Fluid						
			Cells	Globulin	Wassermann			Gold Curve	Mastic Curve
					0.2 C.c.	0.4 C.c.	1.0 C.c.		
B3	Central nervous system, unclassed	3	N. C.	++	4	4	4	5554410000	5554310000
Ax21	Wassermann	4	N. C.	++	4	4	4	5555555221	5532100000
Ax24	Tabes	4	N. C.	++++	4	4	4	5555542100	5554321000
Bx7	Tabes	4	N. C.	++++	4	4	4	5555532100	5554321000
Cx5	Paresis	4	N. C.	++++	4	4	4	5555555321	5555554321
Cx24*	Central nervous system, unclassed	0	N. C.	++++	4	4	4	5554+310000	5554321000
Cx31	Paresis	4	N. C.	+++	4	4	4	5555521000	5555321000
Dx14	Paresis	4	N. C.	++++	4	4	4	5554100000	5555432100
Dx19*	Secondary neurorecurrence	4	N. C.	+++	0	4	4	55442+21100	5553210000
Ex32	Wassermann	4	14	++	4	4	4	5442210000	5555432100
Fx13*	Central nervous system, treated	0	5	++	0	4	4	554+4+4+21000	5553210000
Fx15*	Paresis, treated	0	2	+	0	0	0	5555210000	5554321000
Kx13	Nonsyphilitic ?	0	N. C.	++	0	0	0	5555432200	4321000000
Z1	Paresis	4	N. C.	++++	4	4	4	5555431000	5555432000
Z3	Paresis	4	62	++++	4	4	4	5555432000	5555521000
Z4	Paresis	4	N. C.	+++	4	4	4	5555552100	5555321000
Z5*	Taboparesis	0	20	+++	4	4	4	5554422100	5555532100
Z6*	Wassermann	4	N. C.	++++	4	4	4	5555521000	5555321000
Hx18	Wassermann	4	14	++++	4	4	4	5542210000	5555321000
Hx23	Nonsyphilitic, lethargic encephalitis	0	7	++++	0	0	0	5433210000	554+3211000
Hx20	Central nervous system, unclassed	4	11	+++	0	3	4	5544320000	5554322100
Ix3	Central nervous system, meningovascular	2	26	++++	0	0	0	4442210000	5532200000
Ix5	Paresis	4	20	+++	4	4	4	5555531100	5555421000
Ix7	Central nervous system, vascular	4	90	+++	4	4	4	5444411000	4221000000
Ix10	Tabes	4	40	+++	4	4	4	5554411000	5532100000
Ix11	Tabes	0	20	+++	4	4	4	5543310000	5432100000
Ix25	Central nervous system, unclassed	4	0	+++	3	4	4	5555422100	4321000000
Jx1*	Secondary	0	98	++++	4	4	4	5555430000	5532100000
Jx6	Paresis	4	100	++++	4	4	4	5555554320	5555321000
Jx24*	Central nervous system, asymptomatic, treated	0	61	++++	4	4	4	4+4+44432+200	5555321000

* An asterisk after a case number signifies that the patient had been treated for syphilis by us.

* The globulin test was performed with Pandy's reagent. The Wassermann reactions were made with both plain alcoholic and 0.2 per cent. cholesterinized antigen.

* In this and the following tables, the diagnoses given are those made on the patient's admission, on the basis of clinical evidence. If neurologic symptoms or signs were absent, the diagnosis is allowed to remain unchanged, even when positive results in the spinal fluid examination show the presence of asymptomatic neurosyphilis.

Gold and Mastic Curves Both Paretic.—Of the thirty patients in the group (Table 1), only one was presumably nonsyphilitic; this was a case which had been diagnosed as lethargic encephalitis. In twenty-three of the twenty-nine syphilitic patients there was definite clinical and serologic evidence of neurosyphilis (paresis 9, tabes 4, unclassed neurosyphilis 10), while the remaining six were diagnosed as asymptomatic neurosyphilis on the basis of blood tests and other spinal fluid abnormalities. A comparison of the gold and mastic curves in this group shows that they are almost parallel. The number of tubes in which complete precipitation occurred is sometimes greater with the gold, sometimes with the mastic test; but a fluid showing a very long or a very short paretic gold curve tends to show a mastic curve of the same general type.

Gold Curve Paretic, Mastic Curve Negative.—Only five patients fall into this group (Table 2). All of these patients were neurosyphilitic, and in all the clinical evidence, or that provided by other tests, showed that in these cases the gold curve is to be relied on rather than the mastic. Only one of the patients gave a positive Wassermann reaction of the spinal fluid, and at least two had normal cell counts.

TABLE 2.—Gold Curve Paretic; Mastic Curve Negative

Case No.	Diagnosis on Admission. Form of Syphilis	Blood Wassermann Reaction	Cerebrospinal Fluid						
			Cells	Globulin	Wassermann			Gold Curve	Mastic Curve
					0.2 C.c.	0.4 C.c.	1.0 C.c.		
C14*	Tertiary	0	17	±	0	0	0	4553221000	2221000000
R4*	Wassermann	3	41	+++	4	4	4	5554210000	2111000000
S8*	Tertiary	4	N. C.	±	0	0	0	4442100000	2221100000
1x24	Central nervous system, unclassed	4	0	+++	0	0	0	5554310000	2110000000
Ix29	Central nervous system, vascular	?	4	+	0	0	0	5443310000	2210000000

Gold Syphilitic Zone, Mastic Negative.—There were twelve patients in this group (Table 3), two of whom were definitely nonsyphilitic (one a normal person, one a case of multiple sclerosis). There was confirmatory evidence of damage to the central nervous system in the fluid Wassermann reaction of only two patients, although in three others the globulin test was strongly positive. One of the patients with a positive fluid Wassermann reaction showed a persistently positive blood Wassermann reaction as the only clinical suggestion of neurosyphilis. In the other there was no clinical suspicion of central nervous system invasion. An additional patient had neurosyphilis which had been consistently treated. It is possible that the syphilitic zone gold curve in some members of this group may be explained if the gold test was performed three or four days after the spinal puncture. The fluid is usually not collected in sterile tubes, and bacterial contamination may play some part in the production of gold curves of this type. The presence of the curve in a normal patient in whom syphilis can be excluded lends support to some such explanation.

Gold Syphilitic Zone, Mastic Paretic.—A comparison of this group with the last shows that in thirty-one instances in which a syphilitic zone gold curve was obtained, the mastic test was negative twelve times and paretic in type nineteen times. As stated in the foregoing, we have observed a syphilitic zone mastic curve only once. This was in a patient with cord tumor and xanthochromia. The spinal fluid showed no cells, a + + + + globulin reaction, a negative Wassermann test with all dilutions, a gold curve of the meningitic type reading 0001122411, and a mastic curve which read 224^{+}5532100.

In fifteen of the nineteen patients in this group (Table 4) there was definite clinical evidence of neurosyphilis; and in three of the remaining four the other tests in the fluid substantiated the diagnosis of neurosyphilis. The exception proved to be a case of lethargic encephalitis. In general, the paretic mastic curves obtained here were of a shorter type than those in the group in which the gold curve was also paretic.

TABLE 3.—Gold Curve Syphilitic Zone, Mastic Negative

Case No.	Diagnosis on Admission. Form of Syphilis	Blood Wassermann Reaction	Cerebrospinal Fluid						
			Cells	Globulin	Wassermann			Gold Curve	Mastic Curve
					0.2 C.c.	0.4 C.c.	1.0 C.c.		
H6*	Wassermann	1	N. C.	++	0	0	0	2321000000	2210000000
W11*	Wassermann	2	13	±	1	2	3	1123000000	2211200000
Ax4*	Wassermann	0	N. C.	0	0	0	0	223^{+}3^{+}110000	2211100000
Bx15*	Secondary	4	N. C.	+	1	4	4	1123110000	2222100000
Dx4*	Wassermann	0	N. C.	±	0	0	0	2355110000	2100000000
Dx5*	Secondary	0	N. C.	+++	0	0	0	2233100000	2220000000
Dx16*	Wassermann	4	N. C.	++++	0	0	0	2343100000	2211000000
Ex19*	Central nervous system	0	0	+	0	0	0	1233332000	2210000000
Ix1*	Wassermann	0	2	+	0	0	0	1233332000	1000000000
Ix6	Nonsyphilitic, normal	0	0	0	0	0	0	2332200000	1000000000
Ax17*	Secondary	0	N. C.	++	0	0	0	2445522100	2111000000
Jx28	Nonsyphilitic, multiple sclerosis	0	2	+	0	0	0	12332^{+}00000	2210000000

These results permit the conclusions that a paretic type of mastic curve is even less significant of paretic neurosyphilis than a paretic gold curve; that it is commonly obtained in other types of neurosyphilis; and that it may be found, as may a similar gold curve, in nonsyphilitic neurologic diseases. In only two patients in this group was even a preliminary diagnosis of paresis permissible. However, in our opinion, it must be accepted that a paretic mastic test obtained in a syphilitic patient is indicative of definite damage to the central nervous system.

Gold Curve Negative, Mastic Paretic.—The fact that seventeen of the total number of patients studied have shown colloidal tests of this type lends support to the argument that the mastic test should occupy a more prominent place in the routine examination of the cerebrospinal fluid. Inspection of the data regarding these seventeen patients (Table 5) shows that in eleven there was clinical evidence sufficient to support a diagnosis of neurosyphilis; in eleven the Wassermann test of the spinal fluid was also positive. Only three patients showed neither definite clinical nor other serologic evidence of central nervous system invasion.

Mastic Curve "3", Showing Moderate Precipitation in the First Few Tubes; Gold Curve of Various Types.—This group, comprising twenty-four cases, is of especial interest because of the borderline type of the mastic curve (Table 6). They were divided clinically as follows: primary syphilis, 1; secondary, 5; tertiary, 5; latent, 1; Wassermann positive, 7; cerebrospinal syphilis, 4; and nonsyphilitic, 1. Eighteen had received antisyphilitic treatment prior to the examination of the fluid. In only three instances was the Wassermann test of the fluid positive in any dilution. In one of these (P3) the gold curve was paretic in type; in another case (GX1) it was negative, although the globulin reaction was ++++; and in a third (GX22) it was of the syphilitic zone type. In one case (AX7) the gold test gave a high syphilitic zone curve; the clinical diagnosis was neurosyphilis unclassed. In a case (CX18) with vascular neurosyphilis, the gold curve was also of the syphilitic zone type. One patient (DX9) was nonsyphilitic and had lethargic encephalitis; and seven showed no clinical or other

TABLE 4.—Gold Curve Syphilitic Zone, Mastic Curve Paretic

Case No.	Diagnosis on Admission. Form of Syphilis	Blood Wassermann Reaction	Cerebrospinal Fluid						
			Cells	Globulin	Wassermann			Gold Curve	Mastic Curve
					0.2 C.c.	0.4 C.c.	1.0 C.c.		
C4*	Wassermann	0	8	+	0	0	0	0113110000	4322111000
Ax26*	Central nervous system, meningitis	0	N. C.	++	0	0	0	2223222100	5543100000
Bx29*	Secondary	0	N. C.	++	0	0	1	1112333000	5432110000
Cx8*	Central nervous system, meningeal	0	12	++	0	3	4	2222411000	5310000000
Cx17	Paresis	4	N. C.	++++	4	4	4	1123221000	5554321000
Cx22*	Central nervous system, meningeal	0	N. C.	++	0	0	0	3345210000	5543210000
Ex14*	Wassermann	4	943	++++	4	4	4	0123330000	5555322100
Ex20	Unclassed (encephalitis ?)	0	2	+++	0	0	0	2244441110	5543211000
Ex28*	Wassermann	0	N. C.	+	1	3	4	1232221000	4321000000
Fx10*	Secondary	0	8	++	0	1	4	2+3+32+110000	5543200000
Fx23	Central nervous system, unclassed	0	8	++++	4	4	4	2223341000	5554321000
Gx6	Central nervous system, unclassed	4	0	+++	0	2	4	1223321000	4322100000
Gx11	Tertiary	4	26	+++	0	0	0	1133211000	4432210000
Gx23*	Tabes	0	5	++++	0	0	4	2244210000	5543210000
Gx28	Central nervous system, 8th nerve	0	6	++++	2	3	4	2332100000	5542210000
Gx31	Wassermann	4	102	++++	4	4	4	1123210000	554+2100000
Hx12	Paresis	4	73	++++	4	4	4	2441111000	4455553210
Hx29*	Central nervous system, unclassed	0	35	++++	0	2	4	3333421000	5552221000
Z2	Tabes	4	N. C.	++++	2	4	4	2+443100000	5554321000

serologic evidence of neurosyphilis, rendering the value of the mastic test doubtful in those cases. In the remaining eleven patients, however, there were either clinical signs strongly suggesting neurosyphilis, or a definitely abnormal cell count and globulin content, or both. To sum up: In seventeen of the twenty-four cases in this group, other evidence of central nervous system invasion in addition to the borderline mastic test was present, while in seven cases such confirmatory evidence was absent. This leads to the conclusion that a mastic test of this type must be interpreted only in connection with the other clinical and serologic findings, but that in general it may be considered as a definite abnormality. It is of interest that in only four fluids showing this type of mastic curve was the gold curve abnormal. Perhaps changes may occur in the mastic test before they appear in the gold, and in treated neurosyphilis it may be that the mastic test is rendered negative more slowly than is the gold. Further study of this type of reaction is necessary before its interpretation can be safely determined.

TABLE 5.—Gold Curve Negative, Mastic Curve Paretic

Case No.	Diagnosis on Admission. Form of Syphilis	Blood Wassermann Reaction	Cerebrospinal Fluid						
			Cells	Globulin	Wassermann			Gold Curve	Mastic Curve
					0.2 C.c.	0.4 C.c.	1.0 C.c.		
B19	Wassermann	4	7	±	4	4	4	1222100000	5543210000
M10	Central nervous system, meningeal	0	N. C.	++	0	2	4	1112100000	5543100000
Ax15*	Tabes	0	N. C.	+	4	4	4	0112221100	4553110000
Cx3	Congenital	4	N. C.	++++	4	4	4	0112211100	5553321000
Cx23*	Secondary	0	N. C.	±	0	0	0	0122100000	4322100000
Dx8*	Primary	4	N. C.	±	0	0	0	1111000000	4321000000
Dx28*	Secondary, neurorecurrence	0	N. C.	++	0	4	4	0111110000	5422100000
Ex4	Tertiary	4	9	0	0	0	0	0111110000	4321000000
Ex18*	Tabes	0	2	++++	0	3	4	1222211100	35554+21000
Ex31	Central nervous system	4	16	+++	4	4	4	1222210000	5543221000
Fx16*	Wassermann	0	2	+	0	0	0	1112210000	4321100000
Fx26	Tabes	0	2	++++	0	1	4	0112110000	4332100000
Hx5	Wassermann	4	65	++++	1	4	4	2211000000	5543210000
Hx6	Central nervous system, vascular	?	23	+++	0	0	0	1112221000	4553210000
Ix14*	Central nervous system, unclassed	4	4	++	0	4	4	1222110000	4322000000
Ix17	Secondary	4	14	+++	4	4	4	2222200000	4322000000
Ix18	Central nervous system (?), Bell's palsy	0	13	++	0	0	0	0001100000	4322000000

DISCUSSION

A general survey of the whole material shows that in 108 cases in which abnormalities appeared in one or both colloidal tests, there was complete agreement (both curves paretic in type) only thirty times. There were seventeen instances in which the gold test was positive (five paretic, twelve syphilitic zone) when the mastic test was negative. At least a few of the gold curves of the latter type may be questioned because of possible bacterial contamination of the fluid. On the other hand, in seventeen cases the mastic curve was paretic when the gold was negative, and in all of these cases it appeared to be more closely in agreement with the clinical and serologic evidence provided. In the group of twenty-four borderline mastic curves, the evidence appears to favor the gold four times, and the mastic thirteen times, while in seven instances the result is doubtful. We must emphasize once more the relation of the paretic curves of both colloids to paresis. So far as the gold curve is concerned, practically all cases of paresis show a paretic curve, but this type of curve is frequently found in other types of neurosyphilitic involvement. A paretic type of mastic curve also appears in most cases of paresis, but it is found even more frequently than the gold in other neurosyphilitic diseases. A diagnosis of paresis is therefore never permissible solely on the basis of a paretic mastic curve.

TABLE 6.—Gold Curve of Various Types; Mastic "3" Curve

Case No.	Diagnosis on Admission. Form of Syphilis	Blood Wassermann Reaction	Cerebrospinal Fluid						
			Cells	Globulin	Wassermann			Gold Curve	Mastic Curve
					0.2 C.c.	0.4 C.c.	1.0 C.c.		
P3*	Wassermann	0	N. C.	++	2	4	4	5555431000	3322110000
R5*	Tertiary	0	N. C.	0	0	0	0	1121000000	3221100000
S10*	Wassermann	4	N. C.	+	0	0	0	0112100000	3221110000
Ax5*	Tertiary	4	N. C.	±	0	0	0	1222000000	3222111000
Ax7	Central nervous system, unclassed	0	N. C.	+	0	0	0	111254$^+$5100	3322111000
Cx7*	Tertiary	0	N. C.	±	0	0	0	0012211000	3332110000
Cx18	Central nervous system, vascular	4	N. C.	+	0	0	0	0123111000	3221000000
Cx19*	Central nervous system	0	13	+++	0	0	0	0011110000	3221000000
Cx21*	Secondary	0	N. C.	0	0	0	0	0002200000	3210000000
Cx25	Latent	0	N. C.	0	0	0	0	1121100000	3210000000
Cx26*	Wassermann	0	N. C.	0	0	0	0	112$^+$1100000	3221000000
Cx28*	Tertiary	3	N. C.	+	0	0	0	0001000000	3221100000
Dx9	Nonsyphilitic, lethargic encephalitis	0	N. C.	+	0	0	0	1221000000	3210000000
Dx31*	Wassermann	4	8	++	0	0	0	0111100000	3210000000
Ex1*	Wassermann	4	5	++	0	0	0	1121000000	3210000000
Ex13*	Wassermann	0	3	++	0	0	0	0112000000	3221000000
Fx8*	Secondary	0	2	+	0	0	0	1211000000	3221000000
Fx12*	Secondary	0	2	++	0	0	0	11111$^+$00000	3222000000
Fx14*	Primary	0	15	++	0	0	0	0011110000	3222000000
Gx1*	Secondary	0	3	+++	0	0	4	1111110000	3221000000
Gx3	Tertiary	4	3	±	0	0	0	111$^+$1$^+$1$^+$10000	3221000000
Gx5	Wassermann	4	1	+	0	0	0	0111111000	3211000000
Gx22	Central nervous system, vascular	4	42	++	1	4	4	23$^+$3$^+$1100000	3221000000
Ix4*	Secondary	0	3	+	0	0	0	1110000000	3221000000

We have no explanation to offer for our failure to obtain mastic curves of the syphilitic zone type, unless, as suggested by Stanton,[3] variations in the lot of mastic used is responsible. We have employed four lots of mastic from different sources, with almost equally satisfactory results in three. One lot was found to react in the usual way but not to a sufficient degree, and was therefore discarded.

We feel, however, that the information provided by the mastic test, while in our hands it does not precisely parallel that afforded by the gold test, is of value in the examination of spinal fluids of syphilitic patients. In a considerable number of cases, it has been found to be positive when the gold test was negative.

CONCLUSION

The results which we have obtained show that there is a fairly close parallelism between the colloidal gold and the colloidal mastic tests; and that when agreement is lacking, the mastic test seems to detect abnormalities more frequently than does the gold. This fact, and the simplicity of performance of the mastic test, lead us to conclude that the test should be an indispensable part of the routine of spinal fluid examinations.

DIAGNOSTIC CRITERIA IN EPIDEMIC ENCEPHALITIS AND ENCEPHALOMYELITIS

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INTRODUCTION

In this disease the variegated symptomatology, the great diversity of syndromes met with, the apparent absence of any constant chronology in the development of the syndromes, and the aping by this malady of almost every well-known and well-defined neurologic syndrome, have been responsible for many mistakes in diagnosis and have caused even some of the best-informed students of the disease almost to despair of creating any sort of order out of the clinical chaos presented. But the multifarious clinical forms are gradually being sorted and placed in a relatively small number of groups, the members of each group approximating more or less closely a certain abstract standard of characterization that is now generally recognized as one of the clinical types of the disease. And when the symptoms and signs that are recognizable by modern methods have been accumulated, and when the sites within the nervous system to which they point have been carefully determined, there is in most cases little difficulty in deciding on a diagnosis.

COLLECTION OF THE DATA ON WHICH THE DIAGNOSIS IS TO BE BASED

The Anamnesis.—This should be exact and detailed.

The prodromal symptoms that have most often been complained of include: headache, slight sore throat, chilly sensations, general weakness, disinclination for exertion, and mild gastro-intestinal upsets.

Onset and Development.—In most cases the onset of the disease is abrupt, but the initial symptoms have varied much in different epidemics. In the first Vienna epidemic there was usually a fore-stage with symptoms of slight meningeal irritation, which was soon followed by somnolence and by ophthalmoplegia. In the 1918 epidemic in Australia, there was an initial stage of excitation with headache and sometimes convulsions, and only several days later did the somnolence, the fever and the signs of paralysis appear. In the cases first observed by Netter in France (1918) symptoms of meningeal irritation were absent, but fever was more common than in Vienna. The symptomatic triad described by Netter consisted of fever, somnolence and ophthalmoplegia externa. English observers (1918) recorded asthenia, malaise, headache, general pains, chills, nausea, anorexia and catarrhal inflammations of the respiratory tract as symptoms of the initial stage, followed in a few days, or sometimes almost at once, by any one of several clinical pictures (somnolence with ophthalmoplegia, bulbar paralysis, catatonic stupor, or, comparatively frequently, a Parkinson-like syndrome). In the United States, initial stages of various sorts have been described. When the onset was sudden, there was often severe pain in the head, fever and delirium, followed by a period of improvement for a few days, after which apathy, somnolence and cerebral nerve paralyses of different sorts or Parkinson-like syndromes developed. When the onset was more insidious, the initial symptoms often consisted of diplopia and slight mental confusion, or, in some cases of neuralgias or of pareses in the domain of one or more of the cerebral nerves; and these patients, too, often showed improvement for a few days only to become somnolent later. Not infrequently there was marked restlessness at night with insomnia before a stage of somnolence was reached. In the Hamburg epidemic of 1918-1919, described by Nonne, such an initial stage as described in the foregoing does not seem to have been marked, the disease frequently having been ushered in by paralyses of the cerebral nerves or by a Parkinson-like syndrome. In the recent epidemic in Italy, Switzerland, Austria and parts of Bohemia (1919-1920) the prodromal phenomena have consisted of anorexia, headache, vague rheumatic pains, sometimes diplopia and slight fever lasting for three or four days, then somnolence and external ophthalmoplegia, but, in addition, certain hyperkinetic phenomena (myoclonia, choreatic disturbances of motility, etc.) have been common. During the past year, hyperkinetic cases have occurred also in America. Thus, T. F. Reilly called attention to rhythmical contractions of the abdominal muscles as a common symptom; Ramsay Hunt has recently described cases of "acute infectious myoclonus multiplex"; Tilney and Howe in their monograph have reported choreiform and myoclonic types; Foster Kennedy has described them also under the caption of "acute infective neuronitis"; and Bassoe, Happ and Blackfan, and others in this country have met with choreiform and myoclonic syndromes. It would seem probable, now, that some of the cases described in France as early as 1917 as "Cruchet's disease" were instances of the hyperkinetic form of epidemic encephalitis.

Of great importance for the early diagnosis is an expert general physical, neurologic and psychiatric examination.

Somnolence.—Of the abnormal mental symptoms, somnolence or pathologic drowsiness is by far the most important as a diagnostic criterion. The peculiarities of this somnolence are now so well known and so different, in most instances, from those of uremic or apoplectic coma that they need not be mentioned further.

Deliria.—The deliria that occur in encephalitis are less characteristic than the somnolence. In certain instances they are indistinguishable from the deliria of other toxic-infectious states (initial and febrile deliria; delirium acutum). Sometimes a delirious state assumes the characters of acute hallucinatory delirium (amentia); often the symptoms closely resemble those of delirium tremens (visual and kinesthetic hallucinations) or those of a Korsakoff's psychosis (disorientation as to time, place and persons, with pseudoreminiscences).

Paralyses.—Paralyses or pareses of the lower motor neurons, most often in those corresponding to the cerebral motor nerves giving rise to ophthalmoplegias, facial paralyses, masticatory paralysis, dysphagia or dysarthria, though occasionally also circumscribed paralyses in the trunk or in the extremities, are among the commonest disturbances of motility. Transitory diplopia, unilateral or bilateral ptosis, accommodation paralysis, sluggish pupils and strabismus are important diagnostic marks. Difficulty in reading may persist long after subsidence of the acute attack.

Spastic States.—Far less often than paralyses have there been spastic states (upper motor neuron lesions), though in a few instances hemiplegias, monoplegias or paraplegias have occurred with corresponding changes in the reflexes (ankle clonus; positive Babinski sign). Sicard (1920) has described a "paraplegic type of encephalitis lethargica."

Myostatic Disturbances.—Much more common than spastic states have been those peculiar alterations in tonus corresponding to parkinsonian rigidity, catatonic stupor and catalepsy—the so-called amyostatic syndromes—due to disturbances in the extra-pyramidal motor innervations.

Hyperkinetic Phenomena.—Signs of motor irritation (hyperkinetic phenomena) are now known to be common in these acute nonsuppurative inflammations of the nervous system. In the first Viennese epidemic hyperkinetic phenomena, other than those due to slight meningeal irritation, were uncommon; but in the later epidemics these syndromes have greatly increased in frequency, and in several they have dominated the clinical picture in a majority of the cases. Myoclonias, myokymias, fascicular twitchings, fibrillary twitchings, epileptiform convulsions general or unilateral, choreiform states and maniacal states appear to have become increasingly common.

Sensory Disturbances.—Certain disturbances of sensation can be utilized as diagnostic criteria, though the sensory side of the clinical picture is, in most cases, far less in evidence than the motor side.

Spontaneous pains—vague rheumatic pains; headache; facial, occipital, cervical, brachial, intercostal and crural neuralgias—have been fairly common and often misleading. In a few cases lancinating pains, like those of tabes dorsalis, have been noted, and in a larger number of cases multiple pains like those of polyneuritis. In the latter instances, the muscles and Valleix points have been tender on pressure. Tenderness of the masseter muscles, a sign observed repeatedly by H. M. Thomas, was demonstrable in one of my cases. In another patient who had suffered from extensive cerebral-nerve paralyses, there had been severe bilateral tic doulereux for the relief of which both trigeminal nerves had been injected with alcohol. Typical root pains are not uncommon. With Cross and Irwin I reported one such case (meningo-encephalo-myelo-neuritis) and Bassoe, of Chicago, has described root pains as a part of the meningoradicular syndrome. Boyd has observed herpes in the area of distribution of root pain. Pardee (1920) has described an acute descending radicular type of epidemic encephalitis with sharp lancinating root pains, paresthesias, muscle spasm, hyperesthesia, delirium and fever.

Anesthesias apparently have been relatively uncommon though they have been encountered in isolated cases in neural, radicular, funicular and thalamic distribution. One of my patients had hemiparesthesia with hyporeflexia of the same side.

Disturbances of the special senses have been reported, especially in the domain of the auditory nerve (cochlear and vestibular divisions), in the form of tinnitus, deafness and dizziness. The olfactory, optic, and gustatory nerves appear to have been rarely involved. The eyegrounds are usually normal, though a low grade of optic neuritis has sometimes been reported (Hiram Woods). Boyd mentions two patients who showed pronounced optic atrophy with marked failure of vision. Patients frequently complain of dimness of vision and of inability to read, but these symptoms, as a rule, have been due, less to involvement of visual conduction paths than to accommodation paralysis. The latter is fairly common and is an important diagnostic mark.

Anomalies of the Reflexes.—In most cases the tendon and superficial reflexes are not markedly altered. In some instances they have been hyperactive and, in a few, Babinski's sign has been present. In other rather rare cases, great diminution or even total abolition of the deep reflexes has been noted. The defensive reflex (reflexe d'automatisme) is often slightly increased (Guillain). Reflexes may change markedly from day to day.

Incoordination.—Disturbances of coordination have not been a marked feature in any of the epidemics, though isolated cases of ataxia, most often of the cerebellar type, have been observed. Aphasia, agraphia and apraxia have not been common. Nystagmoid movements of the eyes were observed in five of seven cases by Hiram Woods (1919), and in two these were the only eye-movement disturbances demonstrable.

Autonomic Disturbances.—In autonomic domains some important disturbances have been noted. Alterations in the pupils and in the pupillary reactions have been found to be exceedingly common, and they are now ranked among the more important diagnostic signs. Inequality of the pupils (anisocoria), irregular-shaped pupils, sluggish pupillary reflexes, definite Argyll Robertson pupil (unilateral or bilateral), and total pupillary rigidity have been encountered. Accommodation-spasm and particularly accommodation paralysis have been common. Spasm of the sphincter iridis muscle has also been seen. W. B. Cadwalader (1920) has reported "bilateral sympathetic ophthalmoplegia." This bilateral Horner syndrome consists of: (1) contraction of the pupil, (2) narrowing of the palpebral fissure, and (3) slight retraction of the eyeball. He thinks the bilateral syndrome must depend on diffuse involvement of the brain stem. Salivation has been a frequent symptom. In some instances it has depended on jaw-drop; in others it has resulted from greatly increased secretion. Tachypnea, tachycardia, esophagospasm, esophageal dilatation, neuro-intestinal disturbance, retention of urine, vasomotor disturbance in the skin, and hyperhidrosis are among the autonomic symptoms that have been most frequently recorded. Trophic disturbances (other than muscular atrophies) most worthy of emphasis are a tendency to rapid emaciation and, in the very severe cases, to decubitus. In a few patients a peculiar swelling of the face suggestive of myxedema has been observed. Herpetic eruptions, particularly herpes labialis, have been common, especially in the initial stage. In a few cases scleroderma has occurred as a sequel. Peculiar exanthems (petechial, roseolar, vesicular) have occasionally been seen, but they appear not to have been common. Whether these represent trophic disturbances, or as would seem more probable, localizations of the virus in the cutaneous capillaries, we do not know.

Cerebrospinal Fluid and Nasopharyngeal Washings.—Much attention has been paid to examination of the cerebrospinal fluid. In the early European epidemics many reported negative findings, and a negative spinal fluid was at first regarded as characteristic. William Boyd, who studied the Winnipeg epidemic, states that "a normal fluid was the rule rather than the exception"; in a number of instances he observed increased pressure, slight pleocytosis and a slightly positive globulin reaction. In the United States, though many negative fluids have been reported, there have been a large number in which slight increase in the cell count (small mononuclears), a positive globulin reaction, and a negative Wassermann reaction have been found. When the cell count is increased it is, as a rule, not markedly increased. Counts of from 10 to 100 are much commoner than larger numbers. The cell count may vary materially from time to time in the single case. Thus, in one of our cases it was 97 on the twenty-first day, 133 on the twenty-sixth day and 33 on the seventy-fifth day. Similar observations have been made by Wegeforth and Ayer and by Marie and Mestrezat (1920). In isolated instances counts of several hundred cells or even of several thousand cells have been made; in these cases great care must be taken to rule out the Heine-Medin disease and tuberculous meningitis. No film forms in the cerebrospinal fluid in epidemic encephalitis. A notable amount of sugar, a sign known as hyperglycorachia, has been described (Netter; Dopter). It is not accompanied by hyperglycemia.

As a rule, the cerebrospinal fluid is colorless, though occasionally it is yellowish as in Froin's syndrome. William Hough and H. H. Kerr of Washington have made one such observation. The fluid may or may not be under increased pressure. An interesting observation, frequently made, has been the temporary disappearance of the somnolence and marked relief of the irritation symptoms after lumbar puncture.

Meningococci, pneumococci, influenza bacilli and tubercle bacilli have been uniformly absent from the cerebrospinal fluid. In Austria and in Italy it is asserted that von Wiesner's *Diplostreptococcus pleomorphus* has sometimes been obtained from the fluid and also from the blood. In this country, Loewe and Strauss report that they have demonstrated their filtrable virus in the cerebrospinal fluid as well as in nasopharyngeal washings, and they regard especially animal inoculation with nasopharyngeal washings as an important diagnostic aid in the more obscure cases. They report that they have grown this filtrable virus by making use of Noguchi's method under anaerobic conditions. Levaditi and Harvier have confirmed several of Loewe and Strauss' experiments.

Leukocytosis.—The leukocyte count of the blood is of relatively little help as a diagnostic criterion. In the acute stage there is usually moderate leukocytosis, but in many of the milder cases the leukocyte count has not been increased. In initial stages suggestive of typhoid fever, however, a polymorphonuclear leukocytosis rather than a leukopenia favors the diagnosis of encephalitis.

Urine.—The urine may be entirely normal, though in the severer cases with high fever there is nearly always a trace of albumin and a few casts.

Acuity.—Most of the patients have presented an acutely developing syndrome, but there are also many subacute cases and some remarkable chronic cases with recrudescences and relapses extending over months.

There have also been a number of peracute cases with foudroyant syndromes (encephalitis siderans) terminating fatally within twenty-four or forty-eight hours from acute intoxication. The symptoms are general rather than focal and appear to be of toxic rather than of inflammatory infiltrative origin. The virus, whatever it is, can not only gradually give rise to local areas of inflammation and infiltration widely disseminated throughout the central and peripheral nervous system, but also to intoxication of the body as a whole, causing parenchymatous degenerations of the nervous system and of the viscera (von Economo). If this conception of the pathogenesis of the disease should prove to be correct, we shall be helped by it to understand some of the peculiarities of the symptomatology.

CLINICAL TYPES OR STANDARDS

Out of the welter of symptoms and signs of this protean disease, clinicians have been busily engaged in setting up clinical types or standard symptom-groups. McNalty in England, Bassoe, House, Tucker, Tilney and Howe and others in America, Netter, Sicard, Guillain and others in France, Reichart, Siemerling, Nonne and Naef in Germany, von Economo and Dimitz in Austria and Sabatini in Italy, are among those who have made notable contributions to classification.

Some classifications are based on the mode of onset and course (peracute, acute, subacute, chronic), some on intensity or severity (fatal, severe, medium, mild, abortive, rudimentary, ambulatory), some on the supposed predominant anatomic localization of the process—hence the terms encephalitis, meningo-encephalitis, mesencephalitis, poliencephalitis superior, poliencephalitis inferior, bulbar myelitis, encephalomyelitis, poliomyelitis, polyneuritis, and meningo-encephalo-myeloneuritis. Another classification is based on the supposed cause of the symptoms (toxic encephalitis, inflammatory infiltrative encephalitis). From the more purely clinical standpoint, based on symptomatology, it is desirable to select certain special forms or syndromes as types, making free use of all the classifications thus far put forward. It may be useful to adopt, for the present, the following dominant clinical types, understanding however, first, that in a single case two or more of these types may be observed, either simultaneously or successively, and, second, that as knowledge grows, it may become desirable to change the number:

1. The somnolent-ophthalmoplegic type (febrile or afebrile).

2. The paralytic (akinetic or hypokinetic) type.

3. The amyostatic type (Parkinson-like and cataleptic syndromes).

4. The hyperkinetic type (myoclonic forms, choreatic forms, epileptic forms).

5. The psychotic type (delirious, maniacal, depressive).

6. The hyperalgesic type (painful forms).

7. The tabetic type (Argyll Robertson pupils with loss of deep reflexes and, sometimes, lancinating pains).

8. The ataxic type.

9. The abortive type (formes frustes; imperfect, rudimentary, and ambulatory forms).

10. The aberrant type (intestinal forms, cutaneous forms, vagal forms, etc.).

The names are brief symbols by means of which we eiptomize in each instance a group of symptomatic details. They are vastly useful for the saving of time and for economy of thought. The occurrence of any one of these syndromes at a time when encephalitis is epidemic, especially when it is associated with signs of infection (fever or leukocytosis), should make one think of the possible existence of the disease.

THE CRITERIA FOR DETERMINING THE SITE OF THE LESIONS IN EPIDEMIC ENCEPHALITIS

Many of the syndromes mentioned are now known to be due definitely to certain focal lesions. These nonsuppurative inflammations are widely disseminated pathologic-anatomic processes, but there are certain sites of predilection, and it is because of this that certain clinical syndromes are prone to occur with especial frequency.

The site of greatest predilection in this really terrible disease is the so-called brain stem and especially the gray matter about the aqueductus cerebri and in the floor of the fourth ventricle. Other sites of predilection include: the corpus striatum, including (*a*) the nucleus lentiformis, made up of the globus pallidus and the putamen, and (*b*) the nucleus candatus; the thalamus and the hypothalamic region; the gray matter of the spinal cord, especially of its anterior horns, and the leptomeninges. But foci may occur in any part of the central nervous axis or of the peripheral nerves, including the ganglions on the sensory roots of the cerebrospinal nerves and the sympathetic system.

The limits of this article will not permit a full discussion of the diagnostic criteria for the localization of the lesions responsible for all the disturbances encountered in the disease, but a few of the more interesting points may be referred to.

The somnolent ophthalmoplegic syndrome points, of course, to the interbrain and the midbrain particularly. The somnolence itself probably depends on lesions of the gray matter about the aqueductus cerebri (centrale Höhlengrau), though just what the mechanism is has been a matter of dispute. C. K. Russel (1920) suggests that slight swelling might obstruct the aqueduct and cause acute hydrocephalus. The ophthalmoplegia directs attention to the nuclei of the third, fourth and sixth nerves. The fleeting signs are probably due to intoxication or edema, the more permanent ones to inflammatory infiltration or to toxic degenerative change.

The akinetic and hypokinetic syndromes (other than ophthalmoplegia) are usually easily localizable, for they also are in the main dependent on lesions of the gray matter containing the nuclei of origin of the corresponding motor nerves, though in some instances, the motor white tracts may be involved or even the roots of the peripheral nerves or these nerves themselves. Facial paralysis, abducens paralysis, and paralysis of the muscles of mastication, point to the pons. When these paralyses exist, the picture is that of poliencephalitis superior acuta. When there is paralysis of the muscles of deglutition (dysphagia) or of the muscles of articulation (dysarthria), often combined with disturbances of respiration and of the circulation (dyspnea, tachypnea, tachycardia), the symptoms point to the motor nuclei of the medulla oblongata. Here we have the well-known picture of poliencephalitis, inferior acuta, or bulbar myelitis. Not infrequently such paralytic phenomena are associated with tinnitus, deafness or dizziness (due to lesions of the cochlear and vestibular components of the nuclei terminales nervi acustici). Paralyses of the spinal nerves point to inflammation of the anterior horns of the spinal cord.

When hemiplegias and monoplegias occur with spastic phenomena and a positive Babinski sign, the lesions are most often located in the cerebrum (cortical, capsular or peduncular). Occasionally a pyramidal tract lesion with a positive Babinski sign may be located below the cerebrum (in the pons, the medulla oblongata or the spinal cord). Paraplegia in this disease is most often due to a transverse lesion of the cord, though it may occasionally depend on extensive bilateral cerebral lesions.

The amyostatic syndromes (including the Parkinson-like syndrome and cataleptic rigidity) are most interesting as localizing guides. The picture of paralysis agitans sine agitatione is of frequent occurrence in epidemic encephalitis, and cataleptic rigidity is by no means uncommon. The studies of S. A. K. Wilson, of von Strümpell, of Ramsay Hunt and of others have made it probable that these myostatic disturbances depend on injury to the corpus striatum and especially to the globus pallidus of the nucleus lentiformis.

The several hyperkinetic syndromes may also be valued to a certain extent for localizing purposes. The motor irritation of the initial stage may depend on the general intoxication or on meningeal irritation. Fibrillary and fascicular twitchings point to the cell bodies of the lower motor neurons. Rhythmic myoclonic contractions probably point to supranuclear irritation, though it is possible that some of the myoclonias depend on direct or reflex irritation of the lower motor neurons. Hamill (1920) assumes that the bilaterally symmetrical rhythmical movements point to some mechanism or center in the medulla oblongata. His tracings indicated that the clonic contractions of muscles that come into play in forced respiration had a definite relation to the respiratory rhythm. The rate of twitching equaled the respiratory rate or was double this rate. Choreatic disturbances of motility point to the encephalon, probably to the diencephalon rather than to the telencephalon, judging from what we know of the posthemiplegic choreas and of the thalamic syndrome. It is conceivable that a chorea may be due to diffuse intoxication of the encephalon rather than to localized lesions in small areas, though of this we are not certain. It is possible too that myoclonic syndromes, like choreatic and athetotic disturbances of motility, may depend on the removal of inhibitory influences through injury to their centers of origin. Epileptiform seizures, especially when of the jacksonian type, point to irritation of the cortex cerebri.

The psychotic types doubtless depend on diffuse toxic disturbances or multiple foci of infiltration in the telencephalon, but we know too little regarding the localization of psychic functions to permit a more definite statement. When abnormal mental states persist after the initial stage, one can be tolerably sure that a telencephalitic process or a meningo-encephalitic process exists. This would seem especially probable in patients who exhibit pronounced amnesias, presenting the picture of senile dementia or that of dementia paralytica.

To a certain extent the hyperalgesic states can also be utilized for purposes of localization. Multiple pains of neural and radicular distribution point to multiple neuritis or radiculitis. Typical zone-like pains extending transversely around the trunk, or vertically along the extremities, point to their respective nerve roots (radiculitis) or to foci in the posterior horns of the spinal cord (poliomyelitis posterior). Hemi-anesthesia with violent persistent pains on the anesthetic side, especially with other components of the thalamic syndrome described by Déjerine and Roussy, point to the ventrolateral region of the contralateral thalamus. Howe of New York has reported an example of the thalamic syndrome caused by epidemic encephalitis.

Visual or auditory hallucinations point to irritations of the cuneus or of the first temporal gyrus, respectively, though irritative lesions in the visual and auditory corticopetal conduction paths might be responsible.

Ataxia of the cerebellar type, with defective coordination of agonists, antagonists and synergists, points to the cerebellum, or to the paths connecting the cerebellum with other centers (vestibular nuclei, red nuclei, etc.). Hemi-ataxia can result from injury to the cerebello-thalamic or rubrothalamic paths on one side, or it may follow on lesions of the lemniscus medialis.

The interesting tabetic syndromes now being occasionally reported probably point to an intoxication, injuring the posterior funiculi of the spinal cord and the light-reflex arc somewhere in the midbrain.

Retention of urine and other autonomic disturbances give opportunity for interesting localizing speculation, but we are as yet poorly informed regarding their anatomic basis. Much of what I have said of the localization of the encephalomyelitic foci is based on deductions from our general knowledge of localizing diagnosis rather than on actual pathologic-anatomic studies of necropsy materials from cases of encephalitis that have been well studied clinically.

A fair classification is possible based on the sites of the toxic-infectious processes in encephalitis. The term encephalitis would include: (1) all the inflammatory processes from the telencephalon to the mesencephalon inclusive (telencephalitis, diencephalitis, mesencephalitis), these together making up cerebral encephalitis, and, in addition, (2) all the inflammations of the rhombencephalon (pons and medulla oblongata), including pontile encephalitis and bulbar myelitis. Under the term myelitis would be included all the inflammations that occur in the spinal cord (poliomyelitis anterior, poliomyelitis posterior, myelitis transversa, and myelitis funicularis). Under neuritis could be included the inflammations that occur in the extracentral portions of the cerebral and spinal nerves (radiculitis, peripheral neuritis). Under meningitis, or rather leptomeningitis, would be included patchy inflammations of the soft meninges. Various combinations of these different local inflammations occur in a single case. In some instances it would seem legitimate to speak even of a meningo-encephalo-myelo-neuritis.

DIFFERENTIATION OF EPIDEMIC ENCEPHALITIS FROM SIMULATING INFECTIONS AND INTOXICATIONS

Among the infectious processes with which epidemic encephalitis is likely to be confused stand out prominently: (1) meningitis, (2) influenza and grippal infections, (3) the Heine-Medin disease, and (4) multiple neuritis, especially the infectious form. Confusion may also occur, though less frequently, with (5) typhoid fever, (6) mumps, (7) infectious arthritis or myositis, (8) tetanus, or (9) hydrophobia.

Here, too, should be considered the differentiation from (10) forms of encephalitis, myelitis or encephalomyelitis other than those due to the specific virus that must be responsible for the present epidemic.

Among intoxications that may closely resemble epidemic encephalitis with somnolence may be mentioned: (1) uremia, (2) acidosis, (3) cholemia, (4) drug intoxications, and (5) botulism.

Meningitis.—Early or late in the course of epidemic encephalitis, symptoms of meningeal irritation (general hyperesthesia, rigidity or retraction of the neck, convulsive seizures, photophobia, pupillary disturbances, eye-muscle paralyses, Kernig's sign) not infrequently occur. Indeed, the leptomeninges are now known to show patchy infiltration with small mononuclear cells and sometimes a few polymorphonuclear cells. One of the surprises in these cases, however, is the paucity of changes in the cerebrospinal fluid as compared with the pathologic-histologic findings and sometimes with the clinical phenomena. In many cases, even in those exhibiting signs of meningeal irritation, the spinal fluid may be quite normal, or may show only a faintly positive globulin test or a little increase of sugar. In a certain proportion of the cases, however, the cell count is a little increased, the cells numbering from 10 to 50, chiefly small mononuclears, and the globulin test is distinctly positive. In rare instances a high cell count is encountered. The fluid may or may not be under increased pressure. No film forms in it. The fluid as a rule is colorless, though it may be tinged slightly yellow, or may contain now and then a few red corpuscles.

In epidemic cerebrospinal meningitis the spinal fluid nearly always differentiates the condition from epidemic encephalitis. The fluid is distinctly turbid; there is marked pleocytosis with predominance of polymorphonuclear neutrophils, and meningococci are present in stained smears, either free or inside the cells.

In the forms of purulent meningitis other than epidemic cerebrospinal meningitis, the fluid is turbid; there are many pus cells, and the causal bacteria (pneumococci, streptococci, influenza bacilli, typhoid bacilli) are usually demonstrable in smears, cultures, or by animal experiment.

In the meningitic form of the Heine-Medin disease, the cell count in the cerebrospinal fluid is usually much higher than in epidemic encephalitis.

Mistakes in diagnosis most often occur in the differentiation between epidemic encephalitis and tuberculous meningitis. A number of cases have been diagnosed as epidemic encephalitis only to turn out to be tuberculous meningitis, and the reverse has also been true. Interesting discussions of this confusion are to be found in the papers of Loygue (1919) and of Pirie (1919). The onset is usually more acute in epidemic encephalitis than in tuberculous meningitis, but in some cases there is an insidious onset of the encephalitic symptoms in which event one may easily be misled, especially when a young person is attacked. But in tuberculous meningitis the fluid is often (though not always) turbid; the cell count is relatively high, even though small mononuclear elements may predominate; a film usually forms in the fluid, and quite frequently, though not always, tubercle bacilli can be demonstrated in the centrifugate when a number of specimens are studied. Moreover, the rigidity of the neck and Kernig's sign are usually much more pronounced in tuberculous meningitis. The control of diagnosis by necropsy has shown, however, that the two diseases are still occasionally confused even when the most careful studies are made.

Influenza and Grip.—Many patients who develop encephalitis exhibit the signs of the disease during or shortly after suffering from an infection that has been diagnosed sometimes as influenza and sometimes as la grippe. In some of these cases the infection may, from the beginning, have been that of acute epidemic encephalitis; in other cases, undoubtedly influenza or la grippe has preceded the epidemic encephalitis. We know, too, that influenza and grippal infections other than the Spanish influenza are capable of causing encephalitis as a complication. There is every reason to believe that the infectious agent of the grip group of diseases can localize in the meninges and brain and give rise to metastatic encephalitis. The confusion at present is great because many believe that acute epidemic encephalitis itself is merely a nervous form of grip. Though the evidence now is against this, the whole matter is decidedly unsettled.

Heine-Medin Disease.—Aside from the meningitic form of the Heine-Medin disease, there are still other forms that may be mistaken for one or another form of epidemic encephalitis. We know now that the Heine-Medin disease, besides producing poliomyelitis, is capable also of producing multiple neuritis, meningitis, poliencephalitis, cerebritis and cerebellitis. We know also that acute epidemic encephalitis, though most often giving rise to symptoms of poliencephalitis, diencephalitis or cerebritis, may also give rise to characteristic poliomyelitis pictures or polyneuritic syndromes. Indeed, the outbreak in Australia, described by Breinl in 1918, with its hyperkinetic phenomena and poliomyelitic syndromes, was supposed at the time to be a peculiar form of the Heine-Medin disease. George Draper, in his studies of the English outbreak of epidemic encephalitis, kept strongly in mind the possibility that the disease might be due to the virus of the Heine-Medin malady. In the Italian and Austrian epidemics of 1919, poliomyelitic pictures were often encountered. In the United States, too, several observers have described poliomyelitic forms of acute epidemic encephalitis.

Until we are sure of the exact nature of the virus in the Heine-Medin disease on the one hand and in epidemic encephalitis on the other and can determine their presence in patients, a final decision as to the identity or nonidentity of the two viruses cannot be reached. In the meantime, it would seem best to assume that they are two entirely different diseases, even though we concede the possibility that the one virus may be a modification of the other. Loewe and Strauss find that injection into animals of the cerebrospinal fluid from patients suffering from encephalitis will reproduce the disease. This is not true of the Heine-Medin disease. One attack of the Heine-Medin disease appears to yield permanent immunity, and Boyd has observed the occurrence of epidemic encephalitis in one patient who had had poliomyelitis in earlier life. The diagnostic criteria relied on at present are:

1. In the Heine-Medin disease the paralyses are more or less complete, many are permanent and they are usually present from the beginning, whereas in epidemic encephalitis the paralyses are more likely to come on slowly and to involve one muscle or one group of muscles after another, to be partial rather than complete and to be transient, the muscles later usually recovering their power if the patient does not die.

2. Unilateral or asymmetrical paralyses are common in the Heine-Medin disease, whereas bilateral symmetry of the paralyses is more often encountered in encephalitis.

3. The fever is as a rule highest before the paralysis occurs in the Heine-Medin disease; in encephalitis the fever is usually slight and may be more marked after paralysis has occurred.

4. In the Heine-Medin disease leukocytosis is more marked than in epidemic encephalitis.

5. The cell count in the cerebrospinal fluid is much higher in the Heine-Medin disease.

6. Children and adolescents are more often affected by the Heine-Medin disease than persons of other ages; epidemic encephalitis attacks persons of all ages.

7. The distribution of the typical cases is different in the Heine-Medin disease than in epidemic encephalitis when the two maladies are epidemic at the same time in a given country.

8. In epidemics of the Heine-Medin disease the encephalitic and bulbar-paralytic types are relatively uncommon; in epidemics of lethargic encephalitis the poliomyelitic syndromes are relatively uncommon.

Multiple Neuritis.—There is some evidence that the peripheral nerves may actually be involved in some cases of encephalitis. It is rare, however, even in these cases that symptoms and signs pointing to involvement of the nerve centers are lacking. Furthermore, the paralyses that occur in encephalitis differ in their distribution from those of multiple neuritis. The chronologic sequence is also different, since in encephalitis it is common to have first eye-muscle paralyses, later facial paralysis and masticatory paralysis, then bulbar paralysis and lastly paralyses of the extremities; the reverse order occasionally occurs, but the former is the rule. It would be rare to see such a sequence of events in multiple neuritis. Moreover, ophthalmoplegia externa and sphincter disturbances (bladder and rectum), common enough in encephalitis, are rare in neuritis.

Encephalitis in which there are accommodation paralysis and paralysis of the muscles of deglutition, so that fluids return through the nose, may excite suspicion of diphtheritic paralysis, but in the latter condition evidences of pahryngeal, nasal or laryngeal diphtheria should be present; smears or cultures should yield the Klebs-Loeffler bacillus, and the glands at the angle of the jaw should be enlarged.

Typhoid Fever.—When epidemic encephalitis broke out in this country there were a number of cases supposed to be typhoid. A similar confusion has been reported in other countries. However, a negative blood culture in the first week, later, negative Widal reaction, leukocytosis instead of leukopenia, absence of acute splenic tumor and roseola, presence of paralyses and of hyperkinetic phenomena will exclude typhoid.

Mumps.—It is known that ordinary mumps may be complicated by an encephalitis and that acute parotitis may complicate epidemic encephalitis, since several examples of this have been reported by Lesné and Langle (1920). One observer in this country has described cases presenting the clinical picture of acute epidemic encephalitis (without any complicating parotitis) as mumps-encephalitis because they occurred coincidently with an epidemic of mumps.

Certain clinical features of mumps are helpful in the differential diagnosis from epidemic encephalitis complicated by parotitis. Thus, mumps is extremely contagious from person to person, whereas the contagiosity of epidemic encephalitis seems to be slight. Again, mumps does not occur a second time in patients who have once had the disease, whereas a majority of the patients, who suffer from epidemic encephalitis have perhaps earlier in life suffered from mumps. Pathologic drowsiness, ophthalmoplegia and other paralyses do not occur in mumps unless the mumps be complicated by encephalitis.

Infectious Arthritis and Myositis.—In some of the milder cases of encephalitis the symptoms chiefly complained of are pains in the neck, shoulders and back, or in the extremities, sometimes leading one to think of infectious arthritis or myositis. Careful clinical and roentgen-ray examination, consideration of the mode of onset, a history of pathologic drowsiness, the discovery of slight ophthalmoplegia, of other paralyses or of pupillary disturbances and examination of the cerebrospinal fluid, will usually differentiate.

Tetanus.—In a few instances the diagnosis of tetanus has been made in cases of epidemic encephalitis. Though in encephalitis paralysis of the jaw muscles is commoner than trismus, irritation of the motor nucleus of the nervus trigeminus in encephalitis can give rise to definite trismus. If this be associated with marked rigidity of the neck and spine, one could easily be misled. But in tetanus there is usually (1) a history of a contaminated penetrating wound; (2) fever is not present at first and does not appear until just before death, whereas in encephalitis elevation of temperature is not infrequent at the beginning; and (3) pathologic drowsiness and ophthalmoplegias are absent.

Hydrophobia.—In isolated instances epidemic encephalitis has been confused with hydrophobia or rabies, since in both there may be a hyperkinetic early stage followed by a paralytic stage (with paralysis of the face, tongue, eye muscles and extremities). But rabies is preceded by a history of bite by a dog or other animal, and it is customary when a human being has been so bitten to determine by inoculation of a rabbit or by examination for the Negri bodies or for lesions of Van Gehuchten and Nelis, whether or not rabies actually exists in the animal. Epidemic encephalitis might of course develop in a person who had been bitten by an animal, in which event the differential diagnosis might be difficult.

Forms of Encephalitis, Myelitis and Encephalomyelitis Other Than the Epidemic or Lethargic Form.—Epidemics of encephalitis and of encephalomyelitis have undoubtedly occurred before, but whether they were due to the same virus as the epidemic encephalitis now prevailing is not known. Some investigators appear to believe that the disease now prevailing is entirely new. Others, including myself, think it probable that we are not dealing with a new disease, but with one that caused epidemics in earlier times. Sporadic cases of encephalitis have long been known to occur. That there are many forms of encephalitis due to different viruses (bacterial and other), there can be no doubt. Can we distinguish these other forms of encephalitis and encephalomyelitis from the acute epidemic or lethargic encephalitis now prevailing?

The poliencephalitis superior acuta of Wernicke has long been known to be a hemorrhagic inflammation of the pons occurring most often in drunkards. The acute hemorrhagic encephalitis described by Strümpell and later by Leichtenstern, though it caused clinical syndromes closely resembling some of those observed in the present epidemic, seemed more often to give rise to hemiplegia, monoplegia and aphasia through massive involvement of the telencephalon. The lesions, besides being more massive were much more hemorrhagic in character than has been commonly observed in the present epidemic. It does not seem to me, however, at all certain that either Wernicke's encephalitis or the Strümpell-Leichtenstern encephalitis is fundamentally different from the encephalitis now prevailing. They may possibly have been caused by the same virus or by a close relative. Perhaps it is better, provisionally, to regard them as entirely separate diseases. In the meantime, no wholly satisfactory method of clinical differentiation is available.

Uremia.—In several cases of epidemic encephalitis in my own practice, the patients had been sent to me with a probable diagnosis of uremia, on account of the pathologic drowsiness, associated with albumin and casts in the urine and, sometimes, with arterial hypertension. But the coma of uremia is much deeper than that of epidemic encephalitis; the phthalein output is markedly reduced in uremia and the blood nitrogen greatly increased. Moreover, in uremia the cerebrospinal fluid yields negative findings. In epidemic encephalitis the cerebrospinal fluid may, it is true, be normal, but often there is a slight increase in the count of small mononuclear cells and a positive globulin reaction. It is rare, too, in uremia to encounter the types of paralysis, of hyperkinesis and of myostatic disturbance that are common in epidemic encephalitis.

Acidosis.—Diabetic coma with acidosis may occasionally be confounded with epidemic encephalitis, especially if the patient with encephalitis has undergone starvation and has a trace of sugar in the urine (not uncommon). The mistake should, however, rarely be made, for diabetic coma is always, or nearly always, preceded by a long history of diabetes mellitus, whereas the onset of epidemic encephalitis is, as a rule, acute in patients who do not have glycosuria.

Cholemia.—Here, too, confusion will be rare, the history of preceding hepatic disease and the mode of onset of the cerebral symptoms helping in the differentiation.

Drug Intoxications.—Acute veronal poisoning, cocain poisoning and other forms of drug intoxication might at times of prevalence of epidemic encephalitis lead through drowsiness or delirium to diagnostic mistakes. A drug delirium usually sets in abruptly with confusion and hallucinations. Changes in the reflexes, pupillary anomalies and dysarthria are not uncommon.

A case of veronal (barbitol) poisoning simulating epidemic encephalitis has been reported by Hassin and Wien of Chicago (1920). The patient took 30, 60 and 90 grains, respectively, on three occasions, the ingestion being followed by profound sleep from which she could be aroused to talk coherently but sluggishly. She would fall asleep again during conversation. She had fever, diplopia and asthenia. Often a history of drug addiction is available and will call the physician's attention to possible intoxication of this sort.

Alcoholic Psychosis.—Acute alcoholism with its coma, delirium tremens with its dreamy states, abundant hallucinations, tremor, fever and tachycardia or Korsakoff's psychosis with its disorientation as to time, place and persons, and its memory falsifications might in some instances be mistaken for epidemic encephalitis, which is now known to be capable of causing somewhat similar syndromes. Moreover, encephalitis frequently occurs in patients addicted to alcohol.

Botulism.—In the epidemic that prevailed in Vienna in 1917, the patients were at first supposed to be suffering from sausage poisoning, and in the epidemic in England in 1918 many of the cases of encephalitis were first reported as instances of food poisoning or botulism. In this country, too, epidemic encephalitis was occurring at a time when outbreaks of true botulism due to poisoning from ripe olives were occurring.

At the time of the great war, the use of canned foods (meat, fish, lobster, sausage, mushrooms, olives) was greatly increased all over the world, and opportunities for food poisoning were multiplied. Botulism causes bulbar paralysis and thus simulates the pontile and bulbarmyelitic forms of encephalitis. It is not surprising that confusion of botulism with epidemic encephalitis should have occurred.

In botulism, there is usually bilateral ophthalmoplegia of the intrinsic muscles of the eye, and though this may occur also in epidemic encephalitis, ophthalmoplegia externa is much more common. When botulism occurs *Bacillus botulinus* can usually be demonstrated in the food that has caused the poisoning. Moreover, the persons who become ill are members of groups that have partaken of the contaminated food.

DIFFERENTIATION OF EPIDEMIC ENCEPHALITIS FROM SIMULATING STATES DUE TO VASCULAR LESIONS

The vascular lesions most likely to give rise to confusion with epidemic encephalitis are: (1) cerebral hemorrhage, (2) cerebral thrombosis, (3) cerebral embolism, (4) sinus thrombosis, and (5) cerebral atherosclerosis.

Cerebral Hemorrhage (Apoplexy).—Epidemic encephalitis attacking a person with arterial disease may lead to error. A true apoplectiform onset is, however, uncommon in encephalitis. Further, though hemiplegias, monoplegias and aphasias may be due to encephalitis, they have been uncommon clinical findings in the present epidemic. Moreover, at the onset of apoplexy there is no fever, though a little fever may develop later. With cerebral hemorrhage there is as a rule deep coma, so that it is impossible to arouse the patient by external stimuli, a very different condition from the somnolence of epidemic encephalitis.

Hemorrhage into the pons or medulla oblongata occurring in patients past middle life may, however, cause real difficulties in diagnosis, though gradual extension of the paralyses downward from the eye-muscle nuclei, such as is often seen in encephalitis, is rare in mesencephalic or pontile hemorrhage due to rupture of a blood vessel.

Cerebral Thrombosis with Encephalomalacia.—If a patient has atherosclerosis with low blood pressure (decrescent form of atherosclerosis), cerebral softening may be suspected when in reality encephalitis exists. The presence of fever, the mode of onset and the course of the process will usually serve as clues for differentiation.

Cerebral Embolism.—In patients suffering from a mitral lesion, cerebral embolism might be suspected when the symptoms of encephalitis appear. In encephalitis, however, the symptoms appear less abruptly and are rarely so unifocal in nature as in cerebral embolism. In the pontile and bulbar forms of paralysis one should, however, always consider the possibility of occlusion of a branch of the basilar or vertebral artery.

Sinus Thrombosis.—The autochthonous form of sinus thrombosis that is encountered, especially in chlorotic patients, may closely simulate encephalitis. If there are no signs of stasis in the veins of the skull or face, differentiation may be difficult or impossible, especially if the process is acute and soon fatal. A protracted course indicates encephalitis.

Cerebral Atherosclerosis.—In addition to cerebral hemorrhage or thrombosis there are cases of cerebral atherosclerosis without signs of apoplexy or of sudden arterial occlusion in which cerebral symptoms appear (mental deterioration, restlessness, transitory paralyses, transitory aphasias, etc.) that might be confounded with atypical and afebrile encephalitis. Von Economo has described a remittent encephalitis that during life gave symptoms of pseudobulbar paralysis with athetosis; at necropsy lesions of encephalitis (recent and earlier) were demonstrable. Sometimes examination of the cerebrospinal fluid will differentiate, but only too often the fluid is negative in encephalitis.

DIFFERENTIATION OF EPIDEMIC ENCEPHALITIS FROM SIMULATING STATES DUE TO SYPHILITIC OR PARASYPHILITIC PROCESSES

Cerebrospinal syphilis, dementia paralytica and tabes dorsalis are sometimes believed to exist when the patient's condition is due to epidemic encephalitis. Epidemic encephalitis may occur in patients with cerebrospinal syphilis, in which event one can make sure that syphilis exists, but one may have difficulty in proving the coexistence of epidemic encephalitis. These difficulties have been discussed by Jeanselme (1920).

Cerebrospinal Syphilis.—Epidemic encephalitis can give rise to symptoms that may easily be mistaken for a basilar gummatous meningitis (cerebral nerve paralyses) or for syphilitic meningo-arteritis (convulsive seizures, hemiplegia, aphasia, fever), but if the Wassermann reaction in the blood and in the cerebrospinal fluid is entirely negative, syphilis can be ruled out. Moreover, the optic nerves and optic tracts are often involved in syphilis, rarely in epidemic encephalitis. Oscillation of the symptoms may occur in either disease, though it is more pronounced perhaps in syphilis.

Dementia Paralytica.—I have seen two patients who exhibited marked mental deterioration, loss of memory for recent events, facial paresis and trembling, inability to calculate and change in behavior that led even skilled neurologists and psychiatrists to make a probable diagnosis of dementia paralytica, but in both patients the Wassermann reaction was negative both for the blood and cerebrospinal fluid, and a careful anamnesis revealed a period of pathologic drowsiness and of other symptoms that made an encephalitic origin for the symptoms seem probable.

Tabes Dorsalis.—Encephalitic cases are now occurring in which there are sluggish pupils, or even typical Argyll Robertson pupils (unilateral or bilateral) with or without abolition of the deep reflexes and lancinating pains, cases that a few years ago we should have had no hesitation in pronouncing tabes. But the Wassermann reaction in the blood and in the cerebrospinal fluid is negative and there is no syphilitic history. Cases of this sort were described in Munich by Naef in 1918, and in Hamburg, in 1920, by Nonne, and I have observed two instances.

From now on we shall have to be careful in our valuation of the Argyll Robertson pupil, which we have always regarded as of syphilogenous origin. Cases are now being reported in which the Argyll Robertson pupil has remained as a residual long after the other symptoms of encephalitis have cleared up.

DIFFERENTIATION OF EPIDEMIC ENCEPHALITIS FROM SIMULATING SYNDROMES DUE TO INTRACRANIAL NEOPLASM

Epidemic encephalitis may occasionally give rise to clinical pictures that suggest cerebral tumor, cerebellar tumor, pontile tumor or tumor of the cerebellopontile angle. Tumors of the pons, as is well known, may remain latent for a time and then become acutely manifest.

Lethargic encephalitis may, in all probability, cause lesions of the acoustic nerve with tinnitus, deafness and vertiginous attacks that lead to the suspicion of labyrinthine disease, of an acousticus tumor, of a cerebellar tumor or of a circumscribed meningitis serosa in the lateral recess (Bárány syndrome).

Epidemic encephalitis attacking the cerebellum or the cerebellar pathways and giving rise to acute cerebellar ataxia may excite suspicion of a cerebellar tumor. The course of the disease should soon remove doubt. In brain tumor, the symptoms develop gradually, as a rule, and are, on the whole, steadily progressive over a long period. In cerebellar tumor choked disk is a prominent and early symptom, whereas in epidemic encephalitis, choked disk is rarely if ever seen, and even low grades of optic neuritis are not common.

In tumors of the cerebellopontile angle, the exact chronologic development of the symptoms is characteristic and helps to differentiate from the quicker (sometimes simultaneous) involvement of several cerebral nerves in the course of an acute or a subacute encephalitis. Moreover, in encephalitis, fever and other signs of infection usually precede the occurrence of the symptoms that suggest brain tumor.

DIFFERENTIATION OF EPIDEMIC ENCEPHALITIS FROM CERTAIN OTHER NEUROLOGIC AND PSYCHIATRIC SYNDROMES

Myoclonias.—During the past two years myoclonic forms of epidemic encephalitis have been frequently observed. Excellent descriptions have been given by Sicard and Kudelski (1920) and by Boveri. When encephalitis is epidemic, the occurrence of an acutely developing myoclonia will cause little difficulty in diagnosis, but when there is no epidemic a sporadic case of myoclonia would scarcely be suspected to be encephalitis, or if an encephalitic origin were thought of, differentiation from other myoclonic states (for example, progressive paramyoclonus multiplex) might not be easy. Myoclonia of the abdominal muscles, first unilateral, then bilateral, is a valuable encephalitic sign (T. F. Reilly). Myoclonic contractions of the diaphragm may simulate ordinary hiccup, and it is interesting that epidemic hiccup has been reported (Gotti in Italy; Boyd in Manitoba). A myoclonia that is latent can sometimes be induced by placing the limbs in certain positions or by tapping the tendons (P. Sainton).

An interesting example of hemiclonia alternans has recently been reported by Sicard and Kudelski. There was rhythmic clonus of the right arm and of the left side of the face with paralysis of the left nervus oculomotorius.

Chorea.—Many choreiform manifestations have been seen since the hyperkinetic forms of epidemic encephalitis have been prevalent. In Vienna, Dimitz described this form of the disease as "encephalitis choreiformis." Farquhar Buzzard of London (1919) has reported rhythmic movements of choreatic or of athetoid type that set in weeks or months after the attack of encephalitis. In Baltimore, Happ and Blackfan have described choreiform states in association with pathologic behavior and persistent insomnia in young children suffering from encephalitis. In France, Lesné and Langle, Souques and Harvier and Levaditi have reported certain cases of acute febrile chorea that they believe is a manifestation of epidemic encephalitis. Both chorea minor and chorea major have been simulated by encephalitis. Syndromes like those designated as Dubini's disease, Henoch's electric chorea of young children and Bergeron's electric chorea of older children may all be produced by epidemic encephalitis. When encephalitis is epidemic, therefore, its existence must be thought of when, in association with signs of infection, choreatic disturbances of motility are encountered. A careful analysis of each case may help to reveal the primary disease of which the chorea is a manifestation.

Parkinson's Disease.—Parkinson-like syndromes, especially imitating paralysis agitans sine agitatione, have been frequently seen among the encephalitic cases in all countries. The acute development of this amyostatic syndrome (rigidity, characteristic attitudes, facial mask and poverty of movement with or without tremor) has been a striking feature of this type of encephalitis. In some of these cases the cell count in the cerebrospinal fluid has been increased and globulin has been present, but in other cases the cerebrospinal fluid has been entirely negative.

This Parkinson-like picture is, doubtless, like other parkinsonian syndromes, dependent on a disturbance of the extrapyramidal motor innervations due, in all probability, to lesions involving the nucleus lentiformis of the diencephalon, especially the globus pallidus (J. Ramsay Hunt). P. Marie (1920) maintains that the parkinsonian syndromes of encephalitis differ from genuine paralysis agitans, the latter being rare before 40, unassociated with fever, insidious and slow in onset with steady progression, most often accompanied by a characteristic tremor, not accompanied by disturbances of the tongue or of opening the mouth.

Cataleptic or Catatonic States.—In the first English epidemic, many of the cases were described under the name "epidemic stupor" and in other countries cataleptic states, catatonic states and waxy flexibility have been described as part of the symptomatology of many cases. One of my patients, a physician, when first seen, lay like an image, devoid of spontaneous movement; he was unable to swallow; he answered questions with great difficulty and only after a long interval; he was unable to read (probably on account of accommodation paralysis) and had retention of urine and slight leukocytosis. There was general muscular rigidity, the agonists being involved equally with the antagonists, but there was no evidence of spasticity, no Babinski sign. The face was expressionless. The condition gradually passed off, and this man is making a good recovery.

In asylums for the insane especially, the differentiation of this amyostatic syndrome from the catatonic states of dementia praecox and the cataleptic states of hysteria may sometimes be difficult, but a consideration of the mode of onset and of the course will usually clear up the diagnosis, especially when encephalitis is epidemic. E. D. Bond (1920) emphasizes the fact that mild and transient though definite symptoms of epidemic encephalitis may easily be overlooked in hospitals for mental disease when these symptoms occur in patients who are excited, secretive or indifferent.

Myasthenia Gravis.—Several of the patients suffering from encephalitis in this epidemic have at first been thought to be suffering from myasthenia gravis, owing to certain manifestations (profound asthenia, general muscular weakness, and especially weakness of muscles innervated by the motor cerebral nerves, exhibited as ptosis, facial paralysis, dysmasesia, dysarthria and dysphagia). But myasthenia gravis is a rare disease. It is afebrile. Its course is marked, characteristically, by remissions and exacerbations. On electrical examination, the myasthenic reaction of Jolly is obtainable. There is no atrophy and no reaction of degeneration of the paralyzed muscles.

Progressive Central Muscular Atrophy.—Either the spinal or the bulbar form of progressive (central) muscular atrophy may sometimes be simulated by the more subacute forms of epidemic encephalitis. But it is rare in progressive central muscular atrophy to see an onset as acute as is the slowest onset in epidemic encephalitis. Moreover, in true progressive (central) muscular atrophy there is no retrogression of the symptoms, even though there may be temporary arrests, whereas in epidemic encephalitis, if the disease is not soon fatal, recovery may be complete or improvement be marked, some of the atrophied muscles regaining their normal volume and the fascicular and fibrillary twitchings disappearing.

Hysteria and Neurasthenia.—Some of the more obscure forms of epidemic encephalitis have been erroneously supposed to be cases of hysteria or of neurasthenia. The pathologic drowsiness of encephalitis may be confused with hysterical twilight states or hysterical narcolepsy. The residual neuralgias, the restlessness, the insomnia and the disturbed mentality of the convalescent encephalitic may be ascribed to neurasthenia or to psychasthenia. In encephalitis it is exceedingly uncommon not to be able to demonstrate at least some signs of organic disease (pupillary disturbances; partial paralyses of cerebral nerves, alterations in the cerebrospinal fluid, etc.).

CONCLUSIONS

1. On account of the toxic component and of the inflammatory infiltrative component of the disease process, both general and widely disseminated focal manifestations of involvement of the central and peripheral nervous systems may occur in encephalitis and give rise to the most diverse disturbances of motility, of sensation, of coordination, of the reflexes and of the psyche.

2. Despite the enormous number of clinical forms encountered in epidemic encephalitis, there is a marked tendency to the repetition of certain characteristic forms or types, of which the somnolent-ophthalmoplegic, the paralytic, the amyostatic and the hyperkinetic are the commonest.

3. When the disease is epidemic, it can usually, in outspoken cases at least, despite the clinical diversity, be easily recognized, though in abortive, imperfect, rudimentary and aberrant cases great difficulties in diagnosis may be experienced, and doubtless many cases remain entirely unrecognized.

4. The occurrence in a patient of (*a*) pathologic drowsiness (lethargy), (*b*) cerebral nerve paralysis (especially ophthalmoplegia), (*c*) an acutely developing parkinsonian syndrome, (*d*) a cataleptic or a catatonic state, (*e*) myoclonia, (*f*) chorea, (*g*) pupillary disturbances, (*h*) violent neuralgia, (*i*) a poliomyelitic syndrome, (*j*) a peculiar delirium, (*k*) a psychotic state or (*l*) signs of meningeal irritation in times when encephalitis is epidemic, should make one think of the possible existence of the disease.

5. Though epidemic encephalitis may simulate any one of a large number of neurologic and psychiatric syndromes of entirely different origin, the mode of onset, the course, and the results of carefully conducted neurologic and psychiatric examinations (including an examination of the cerebrospinal fluid), will usually yield the diagnostic criteria that suffice for its recognition and differentiation.

PHYSICAL FINDINGS IN THE PSYCHONEUROSES*

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At the present time there are almost as many opinions about the etiology of the psychoneuroses as there are men who think about them. One end of the scale is sounded by those who believe that the neuroses spring entirely from mental repressions and conflicts. The antithesis of this is the absolutely organic theory which holds that psychoneurotic states are produced solely by physical disease. For some time it has been apparent that there is a tendency to depart from these two radical conceptions. Experimental studies and clinical observations show, beyond a reasonable doubt, intimate association between emotions and mental life in general and the physical state. We may still be honestly uncertain as to where to place the starting point of the neurosis: in the body, or in what is popularly known as the mind.

During the two year period, 1918-1920, 260 patients presenting the symptomatic picture of hysteria, neurasthenia, psychasthenia, anxiety states, etc., were examined and treated at the mental and nervous clinic of the Pennsylvania Hospital. Of this number, 140 revealed only minor pathologic changes. In the remaining 120, or 46.1 per cent., there was definite, and to my mind significant, organic disease. Given in the order of their frequency, we found: endocrine dysfunction, tuberculosis (including the pulmonic, intestinal and glandular varieties), syphilis, extensive apical abscesses, organic heart disease usually with beginning decompensation, postinfluenzal states, arteriosclerosis, anemia, combined heart and kidney disease, osteo-arthritis, sinusitis, infected tonsils, extreme visceroptosis, infected pelvic structures, chronic Neisserian infection, nephritis, chronic appendicitis, suppurative otitis media, prostatitis, gastric and duodenal ulcer, beginning gastric carcinoma, lead poisoning, floating kidney and diabetes. There were nineteen instances of ductless gland disturbance involving the thyroid, pituitary, and suprarenal glands; eighteen cases of tuberculosis, and fifteen patients with syphilis. Of the entire 120 cases, eighty-five were symptomatically expressed as neurasthenia, twenty as psychasthenia, twelve as hysteria and three as anxiety neuroses.

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* Read at a Clinical Conference of the Bloomingdale Hospital, White Plains, N.Y., March 30, 1921.

REPORT OF CASES

It may be worth while to summarize a few illustrative case histories.

CASE 1.—A widow, aged 37, with four children, had a combined hysterical and neurasthenic reaction. She complained of intense headache, both frontal and occipital and severe lumbar pain. There were numerous paresthesias. On the external aspect of the left thigh was a large, irregular area of sensory dulness. When this was touched by the bed coverings at night, marked discomfort and insomnia resulted. There were frequent "hysterical-like" paroxysms of weeping, and the patient constantly dwelt on her belief that she was becoming insane. The illness was of one year's duration. Previous to that time she had operated a 5-pound hammer at a steel mill. She had been under medical treatment for eleven months, and the diagnosis was neurasthenia.

The blood pressure was 215 mm. of mercury. The ophthalmologist reported marked sclerosis of the eye vessels. Further study and subsequent improvement under treatment made it evident that the neurosis was largely conditioned by organic changes, of which the unusually high blood pressure was one important manifestation.

CASE 2.—A widow, 28 years old, with four young children, was "nervous," worried, cried frequently and constantly felt a lump in her throat. There were insomnia, headache, "sparks" before the eyes, a numb sensation in the hands, and a feeling in the head as if two close-fitting wheels were "grinding together."

She had a neurotic make-up, and she had had several terrifying experiences. At the age of 12, she discovered her father hanging from the staircase—a suicide. Shortly before this she had witnessed the death of an older sister by drowning. Some years later there was a fire in the factory where she was employed, and the patient became rigid and unconscious. Her husband died of tuberculosis about a year before.

Examination revealed inactive tuberculosis, beginning hyperthyroidism, a badly lacerated perineum, uterus with inflammatory changes, advanced pyorrhea with many apical abscesses, and chronic otitis media with offensive discharge.

The course of treatment was first complete rest in a convalescent home, then the teeth roots were extracted, the ear condition cleared up and the torn pelvic structures repaired. Hand in hand with these reconstructive measures, there were psychotherapeutic treatments. She is now practically well.

CASE 3.—A woman of 30 with an infant was described as a "nervous crank" who "screamed whenever anything went wrong" and "had a mania for going to hospitals and dispensaries." Her reaction was essentially neurasthenic. Fatigue, aches, pains, discomforts, and odd sensations were the burden of her story. There were incipient pulmonary tuberculosis, systolic blood pressure 90, diastolic 52, muscular adynamia, bronzing of the skin and thyroid struma. The progress of the condition confirmed the diagnosis of Addison's disease.

CASE 4.—A woman of 39 presented a pronounced anxiety state with phobias, the most prominent being a fear of dire calamity if she should leave her house except on the most urgent errands. The neurosis had followed the death of her husband a year before. He had been extremely jealous and would leave his work two or three times a day to find out whether his wife was at home. Examination revealed pulmonary tuberculosis, apical abscesses and a femoral hernia.

It is true that this patient presented important psychologic difficulties which needed to be adjusted, but psychotherapy alone would have left an unfinished problem.

Case 5.—A man, 38 years old, was exceedingly timid, and his collection of phobias was increasing by leaps and bounds. He was "nervous" and "shaky," could not eat and feared that he was losing his memory. The patient was a painter. He often had gastro-enteritis with severe abdominal pain and had lost about 30 pounds in weight. There was advanced pyorrhea, and the roentgenogram revealed extensive apical abscesses.

Here, there was apparently no need for psychotherapy. Rest, change of occupation and attention to the teeth have resulted in a speedy recovery.

Case 6.—A woman of 60 presented a neurosis which came on with "a noise in the ears" following her daughter's death. The tinnitus kept her from sleeping. Soon a number of phobias developed, chiefly the fear of being alone and the fear of going to bed. She began to sit up all night. There were constant drawing pains in the head and arms. The otologist reported severe catarrhal otitis media with partial deafness. The roentgenologist found apical abscesses, the laboratory a low grade nephritis, and the internist a visceroptosis. Under treatment this patient is showing marked improvement.

COMMENT

How are we going to regard the organic morbidity of the psychoneuroses? Perhaps, after all, the occurrence of tuberculosis, syphilis, ductless gland disturbance, heart and kidney disease, apical abscesses, etc., is a matter of coincidence, and no relationship at all exists between these diseases and the neurosis. This would seem more plausible if these factors were unusual and infrequent, but when they may be discovered in almost 50 per cent. of our patients, it seems likely that we are dealing with something more than mere chance association.

Students of heredity will be apt to regard this physical inferiority as an expression of so-called psychopathic inheritance. It is, of course, true that this might influence the particular variety of mental reaction. However, if it were solely a question of physical inferiority, one would expect it to take a more uniform type; and further, to assume a more typical clinical syndrome. In our patients twenty-six more or less common diseases were represented, conditions encountered every day in general practice.

I have heard the objection that these cases merely simulate the neuroses and that basically and essentially they are not hysteria, neurasthenia, psychasthenia and anxiety states. If they are not, they certainly are often faithful duplicates. After all, a diagnosis must be made on the symptoms, and the picture of neurasthenia, for instance, is too clear to escape recognition.

May we assume a direct causal relationship? If so, how does the organic disease operate? Dana[1] deplores the emphasis placed on the psyche as the sole etiologic factor in the psychoneuroses. His argument is based on the theory of "synapsis" which implies "breakable points and points of variable resistance in the finer organizations of the brain." Here the traveling nerve impulse meets its highest resistance, and neural phenomena are apt to develop. Paths of conduction may be blocked or made free not only by mental, but also by physical agencies and endocrine disorders, toxemias of various kinds, etc., may all act as etiologic agents. Wechsler[2] found marked signs of dysfunction of the pituitary gland in four typical psychoneurotic patients, of the thyroid gland in three, and of the suprarenal and sex glands in two. Treatment was successful, and the author seeks in "disturbance of the glands of internal secretions a source of rational therapy." Robertson[3] is convinced that chronic bacterial infections are an important element in the etiology of neurasthenia. The nasal passages, the lower respiratory tract, the nasopharynx, the mouth, the fauces, the intestinal and genito-urinary systems were investigated in sixty-six cases. The *B. influenzae,* the pneumococcus, several strains of streptococci and diphtheria bacilli were isolated and cultivated. Immunization produced important therapeutic results. Carver and Dinsley[4] made some interesting observations on healthy soldiers in an "ammunition proof and demolition section" during the course of their ordinary duties. They were well sheltered, and the element of fear was minimal. Various symptoms were noted, including active tremors, increased pulse and respiration rates, and in a few cases, vomiting. Those most affected became less resistant to subsequent exposure. As a result of their experiments and of observations on soldiers, the authors believe that the "indirect concussion" of high explosives produces definite changes in the central nervous system accounting for certain of the symptoms usually ascribed to psychogenic factors.

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[1] Dana, Charles L.: The Somatic Causes of Psychoneuroses, J. A. M. A. **74**:1139 (April 24) 1920.

Obviously it is unfair and dangerous to dismiss the psychoneurotic without adequate examination. Consultations, serologic, biochemical and radiographic studies may all be needed for our difficult problems. Finally, it seems evident that no single system is sufficient for the treatment of the neuroses. When we really do not know it is not safe to assume or to theorize, especially if our hypotheses mean that etiologic possibilities are to be excluded in actual practice.

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[2] Wechsler, F. S.: The Psychoneuroses and Internal Secretions, Neurol. Bull. **2**:199 (May) 1919.

[3] Robertson, W. Ford: The Infective Factors in Some Types of Neurasthenia, J. Ment. Sc. **75**:16 (Jan.) 1919.

[4] Carver, Alfred, and Dinsley, A.: Some Biological Effects Due to High Explosives, Brain **42**:113, Part 2, 1919.

News and Comment

DUTCH SOCIETY OF MENTAL HYGIENE

Headed by Dr. Ariëns Kappers and Dr. B. Brouwer, a society for the scientific study and promotion of nervous and mental therapeutics has been formed in Holland. It includes all the professors in neuropathology and psychiatry in Holland and several pharmacologists. The society is sustained by subscriptions. It publishes a periodical called *Neurotherapeutics,* for which contributions are accepted in all modern languages. Contributors are paid $13 for each sixteen pages of printed matter. The society also invites foreign physicians to deliver lectures on their work, Professor Petrén of Sweden and Prof. F. Plaut of Munich being among recent lecturers. It is expected that Levaditi will lecture to the society in October. In an announcement, physicians are offered membership for the annual fee of 10 florins, life membership for 250 florins or associate membership for 5 florins yearly or a single sum of 100 florins. The society may be addressed through the secretary, Dr. B. Brouwer, Koninginneweg 170, Amsterdam.

FIRST SESSION OF THE FRENCH LEAGUE OF MENTAL HYGIENE

The first session of the French League of Mental Prophylaxis and Hygiene was held in Paris, June 1. Addresses were delivered by the president, Dr. Toulouse, and by Justin Godart who defended the right of the insane to support by the government, by Prof. Jean Lépine who requested the creation of dispensaries for psychopathic patients, and by Dr. F. Williams, director in France of the Rockefeller Foundation. Requests for admittance to membership in this organization should be addressed to the secretary, Dr. Genil-Perrin, Avenue de la Bourdonnais 99, Paris, 7th arrt.

Abstracts from Current Literature

APHASIA: A HISTORICAL REVIEW (HUGHLINGS JACKSON LECTURE FOR 1920). Henry Head, Discussion by Drs. James Collier, James Purves Stewart, Kinnier Wilson and others, Brain **43**:391, 1920.

The Hughlings-Jackson lecture for 1920 by Head is a very complete and interesting historical critical review of the subject of aphasia. The story of the influence of Joseph Gall, Broca, Bouillaud, Trousseau, Jackson, Bastian, Baginsky, Wernicke, Lichtheim, Moutier, Déjerine and others on the conceptions which we have held in the past concerning this subject is interestingly put forth.

Gall, who localized speech in "those portions of the brain which lay in relation with the posterior part of the orbital cavity," . . . "for as a boy he noticed that his companions who had prominent eyes had a gift for languages and a memory for words," is given credit for being the first to take into account the fact that speech and intelligence are probably cortical functions. Mention is made of Broca's studies and of the fact that the brain exhibited by Broca at the Societe d'Antropologie, which served as a basis for Broca's idea of the localization of the speech function, was not sectioned and properly examined anatomically until Pierre Marie did this forty-five years later. The fact that Marie was able to disprove Broca's deductions by his studies of Broca's material is also mentioned. A careful review is made of Jackson's views, who, for the first time, definitely divided healthy language into the (1) intellectual, i. e., the power to convey propositions, and (2) emotional, i. e., the ability to exhibit states of feeling. Jackson believed that these two parts of speech were separated by disease and that in most cases of disease the former only was affected. As Hughlings Jackson said, the question is not how is general mind damaged, but what aspect of mind is damaged in aphasia?

Head treats Wernicke's views, particularly in his discussion of the case of isolated agraphia, with scant respect, and likewise shows little patience with the views and diagrams of Lichtheim and of many others.

Marie referred the aphemia (motor aphasia) of Broca to difficulties in the lenticular zone, while the aphasic side of speech defects (sensory aphasia, auditory aphasia and verbal amnesia) were referred to disturbances in Wernicke's zone. According to Marie, the true aphasias are a disturbance of general intelligence and of special intelligence for language. Head gives some attention also to the controversy which took place between Déjerine and Marie over the latter's views. He speaks of these debates, which at times became very bitter, as purely verbal battles, which would have resulted in some clarification of their points of view had the disputants been familiar with Hughlings Jackson's ideas. Into these Head goes rather minutely and points out again that Jackson was a pioneer in classifying aphasics properly into two great classes; first, those who are speechless or practically so, and second, those who have a plentiful vocabulary but who use words in a wrong way. He agrees with Jackson that there cannot possibly be such a condition as pure agraphia because a man who cannot write spontaneously may be able to copy printed matter in perfect handwriting. "He cannot write voluntarily because he has lost the use of written words in propositions." The fact that such patients can write their names and addresses or those of intimate friends or relatives is ascribed by Jackson and Head to the theory that these things "with time more nearly reached the level of an automatic act." Head emphatically agrees with Jackson that in the majority of cases of aphasia mental images are not affected. As Head says "It is not sufficient to hold up some object and ask the patient to name it . . . his power of responding must be tested by a series of observations in which the same task occurs on two or more occasions and each of the tests must be put before the patient in several different ways . . . so that the choice of any one object (in the tests) recurs three or four times in the course of the series." Head gives a short abstract of a description of his tests which have been described in *Brain*, volume 43, pages 91 to 107. Then follows a summary of Head's views which were also expressed fully in that paper and were abstracted by the reviewer in the ARCHIVES OF NEUROLOGY AND PSYCHIATRY, December, 1920.

Head's ideas might be summarized in his statement that "we should as soon expect a special center for eating as for speech . . . no lesion, however local, can affect speech and speech only. Cerebral injury disturbs certain physiologic processes which subserve the complex acts which we speak of as speech. Any organic injury which produces a disorder of speech disturbs other functions not usually associated with language, or leaves unaffected much that undoubtedly belongs to speech." Thus Head disposes of all the old terms with their underlying conceptions, such as motor and sensory aphasia, apraxia, agraphia, or verbal amnesia. Head ends this summary of his views by saying, "No disorder of speech due to a unilateral lesion of the brain corresponds exclusively to any one of these categories; still less can these hypothetical conditions be associated with limited destruction of any one part of the brain . . . any mental process is liable to suffer which demands for its performance exact comprehension, voluntary recall and perfect expression of symbolic representations."

At this point the reviewer wishes to take the liberty of pointing out that several times in Head's recent writings the statement is made that intelligence as such is something entirely apart from speech and may be unaffected by disease processes causing aphasia.

If Head's views just quoted are true, how can it be possible that any aphasic will not show some disturbance of the intelligence? Before this point can be intelligently discussed every one must have clearly in mind what is meant by the word intelligence. Either one agrees with Marie that there is a special intelligence for speech and that no disturbance of speech resulting in aphasia can possibly occur without some disturbance of the intelligence, or he believes with Head that intelligence is developed only incidentally with speech and that it is possible to damage seriously the speech function without disturbing the intelligence, or he agrees with still others (see Purves Stewart's discussion) who hold tenaciously to the speech center theories and their diagrammatic conceptions. The latter would have us believe that the speech function is more or less sharply localized in various points of the brain and that it is possible to injure these areas with consequent disturbance of speech in which only that function and nothing else is disturbed.

Head's statements just quoted would seem to place him in the same position as Marie and the reviewer who believe that intelligence and speech develop apace and are intimately bound up together in the brain. This is so because the mind which thinks does so only in speech or, as Head calls it, symbolic expression; whether that be spoken or not is immaterial. This on the anatomic side simply means that the entire brain is connected up in an intricate, complex way by means of short and long association paths and that many, if not all, of these pathways must be used in ordinary symbolic expression. It is, therefore, ridiculous to talk of speech centers. There must be in areas of the brain that are fairly well known, which necessarily vary in development and localization with persons, certain places where memories of various kinds are stored. These association areas are connected with each other and with various parts of the brain having other functions by certain pathways which, when interrupted, not only disturb the speech function but necessarily disturb also other functions of the brain which, taken all together, make up general intelligence. As we understand intelligence, it is a combination of all the memories and experiences of the person which are capable of being expressed in thought. As Head well says, "The higher the propositional value of the mental act, the greater difficulty will be present." General intelligence certainly is affected in those cases of Head's which he calls semantic aphasia. His idea is that in these cases there is an inability to recognize the significance of words and names. The reviewer believes that if a person is unable to do this certainly his intelligence generally must be seriously affected.

Dr. James Collier, in his discussion, said that the clinical varieties of aphasia described by Head "correspond to definite stages of dissolution or recovery: neither do they reveal directly the elements out of which language is built up. On the contrary, they show the components into which a highly complex state of psychical processes can be separated by destruction of certain portions of the brain." Collier further said that the "alterations in speech function which result from lesions of the brain are not in terms of the primary elements out of which speech has been evolved, nor do they reveal the elementary basis of the acts of speaking, reading and writing, but that they are in terms of the complete and highly developed function, which fails progressively from its highest to its lowest with increasing degrees of damage."

The reviewer believes this situation is similar to the tearing down of a house, for one can see in the débris the elements out of which the house was built. If the functions of speech and intelligence, which are localized in the cortex of the brain, are destroyed or disturbed by disease or injury, it is difficult to see why we are not justified in concluding that the resultant defects are important portions of the mosaic from which they have been removed, study of which may give important clues as to how intelligence and speech are patterned in the brain. If this is not true, then there is little importance in studying this subject, and the possibility of ever learning how speech and intelligence are developed must be given up as hopeless.

Head then goes on to review his conceptions of the four types of aphasia as classified by him, namely, verbal, syntactical, nominal and semantic aphasia. He, of course, admits that none of these groups is clear cut and that many of the cases merged some of the characteristics of each group; he offers them simply as a means of study and convenient grouping. As a matter of fact, the semantic and nominal groups seem to show a great many points of similarity.

Collier's discussion is chiefly important for the several points on which he differs from Head. He believes in the "existence of afferent paths to the speech region on the auditory side and on the visual side and efferent paths therefrom, by which speech is usually exteriorized, both spoken and written, and in these paths there is surely strict anatomical and physiological separation."

Collier believes that an "isolated involvement is possible if a lesion hits one of these anatomical tracts before it merges with others or breaks up into associated pathways." He disagrees with Head and agrees with Déjerine that a case of pure alexia may occur, and he claims to have seen the lesion demonstrated in the posterior limit of the speech area. While Collier agrees with Head that pure word deafness does not exist, he thinks that pure agraphia and pure motor aphasia may occur. Yet Collier says "there are cases which all of us must have seen in which lack of acceptance of spoken speech stands out in undue contrast with the acceptance of visual speech." He lays great stress on the recognition of speech "by means of the auditory path which allows of analysis of the correctness and fitness of what we have said and of its correction, if faulty." Collier also emphasizes the importance of the feel of speech, i. e., the sensations which accompany the movements of the speech mechanism. He says that when both these things, but especially the auditory immediate consciousness of the correct execution of speech, fail, then confusion must certainly be the result in thought and speech. Collier believes that the visual and auditory speech areas and functions cannot be sharply separated because the "function of visual speech, acquired at a much later age than is auditory speech, tacked on as it were to an already perfect and deeply impressed function of auditory speech, learnt by means of an already well versed auditory speech function, must occupy the same physiologic substratum as does the auditory speech function and is separately localized only in so far as its chief afferent and executive paths are concerned."

Collier agrees with the principle expounded by Head that the alterations in speech function, which result from lesions of the brain, do not reveal the elementary basis of the acts of speaking.

The reviewer has already discussed this point and in further disagreement would point out the fact that a good deal of the physiology, for instance, of automatic associated movements and tremors, and practically all of the knowledge that we have concerning these conditions has been taught us by the study of the pathology of these functions in the basal ganglions and in the midbrain. Why this cannot be so with speech is difficult to understand. Collier agrees with Head that the cerebral cortex concerned with speech functions acts as a whole and not in terms of isolated localized functions. He disagrees with Head's conception of syntactical aphasia and does not believe that this condition can arise from a lesion of the cortex. He thinks that jargon aphasia is a thing quite of itself and is characterized by overaction and volubility. In this condition the intelligence and quickness of apprehension stand out prominently, but the volubility seems to be due to a severance from control and lack of cortical guidance. Collier thinks that this condition is due to a blocking of the auditory afferents. He speaks of two patients who pathologically showed subcortical tumor "undercutting the posterior and mesial aspects of the temporal lobe, comparatively far from the cortex which was in no way involved." Collier says, further, that jargon aphasia is hardly ever encountered in the common lesions of the temporal region . . . "where the temporal cortex is likely to be involved." The reviewer believes also that jargon aphasia is due to a progressive lesion. An extension of the lesion to the temporal cortex causes the jargon aphasia to lessen in volubility and finally to disappear.

The discussion by Sir James Purves Stewart can be summed up in a few words. He sees no reason to change the conception of psychomotor aphasia and psychosensory aphasia and proceeds to discuss six cases of aphasia resulting from craniocerebral wounds (with one exception which was a vascular lesion in a man aged 55). Stewart's cases are not completely given. There is no record of a complete neurologic and aphasic status and nothing is said about the handedness of the patients or of their previous condition and education.

It occurred to the reviewer in reading Stewart's cases and even those which were the basis for Head's studies to ask what tests the observers had made to determine that the injuries were purely limited to one side of the brain. How could they know that there was not more than one lesion on both sides of the brain? Nothing in the reports of Head's or Stewart's cases answers this question. There is a lack of complete neurologic status and certainly a complete lack of pathologic report with which to exclude this important point. Stewart believes that the old conception of psychovisual, psycho-auditory and psychomotor speech centers are justified.

The discussion of Kinnier Wilson follows and seems to be in perfect agreement with our point of view concerning the type of case used for both Dr. Head's and Dr. Stewart's studies. Wilson points out that even Head admits that gunshot wounds "tend to cause greatest damage on the surface of the brain in addition to which there is the factor of commotio in all such patients. The fact that small hemorrhages may catch projection fibers where they converge or diverge is, in my opinion, much less important for the subject of aphasia than that association, i. e., trans-cortical fiber systems should be involved . . . the ideal case is neither the tumor nor the gunshot wound case but that with isolated vascular lesion, the exact position of which there is opportunity to establish postmortem."

Wilson takes exception to Head's interpretation of the difficulties shown by patients with aphasia in imitating movements when confronting the examiner as against the ease with which the same movements are performed when the patient sees them in a mirror, the examiner being in back of him. Wilson does not believe that these difficulties occur because of disturbed verbalization but are due to a disturbance of automatism resulting in dyspraxia or apraxia. It seems to us, however, that Head's interpretation of the difficulty is the true one because, in attempting to imitate movements not commonly used, a certain amount of thought in the form of internal speech is necessary so that one may attend to the matter in hand. Of course, Wilson's interests are along the lines of establishing in apraxia the same sort of diagrammatic fallacies that we have been so long in breaking down in aphasia, and his entire discussion is directed along these lines. He quarrels with Head's grouping of the cases and does not see why it should be necessary to discard our old terms with some of their underlying conceptions. Particularly does he disagree with Head's fourth classification (semantic aphasia). He believes that these cases are really nothing but the agnosias described by Liepmann and others.

The reviewer has on a number of occasions called attention to the fact that the questions of aphasia, apraxia and the intelligence are intimately bound up, one with the other, and any attempt to separate them definitely as functions or "faculties" of the brain will result in artificial classifications and conceptions which run counter to established physiologic, psychologic and anatomic facts. Saying, as Wilson does, that motor aphasia is nothing but an apraxia as applied to speech is a truism. This, however, has not helped to explain to any great extent, in the absence of any sensory or motor paralysis of the organs involved in speech or motion, the mechanism either of disturbances of speech (aphasia) or of volitional movement (apraxia). So, too, to call the sensory aphasias a form of visual or auditory agnosia does not help matters.

Dr. Stanley Barnes in his discussion obviously has failed to read Head's original article carefully for he expresses a desire "to have heard more emphasized by Dr. Head that intellectual faculties were so closely bound up with speech that one must expect them to be represented in the same area of the brain and that any serious defect in speech must mean a serious defect in intellect too." Head has definitely said that the opposite of this statement is true. Barnes, however, believes that "the two functions, speech and intellect, were largely interdependent and were represented in the same area of the brain."

Dr. Barnes' discussion agrees so closely with what the reviewer thinks of this whole subject that to dilate on it would simply be to repeat this statement over and over again in different words.

The psychologic discussion by Dr. James Herbert Parsons sems to be the basis for the same sort of a conception as that expressed so broadly by Dr. Barnes and in a more narrow sense by Head. Parsons sketches rather interestingly the psychology of the development of "meaning" and shows how intricate a process learning is and how impossible it must be to have the function of speech and thought localized in any circumscribed areas of the brain. The process is one of delicate and intricate association, and our thought and speech are learned in a variety of ways through numerous efferent sensory pathways with boundless primary and secondary associations resulting in a complicated function.

Head in reply could not understand why Dr. Collier wished to keep up terms used in describing aphasia which were physiologically, anatomically and psychologically impossible. For these reasons Head had sought to replace them by something more fundamentally correct.

In describing syntactical aphasia Head says that the important thing is that such patients lose the regulating power over the rhythm and balance of what is said because speech is not recognized in a critical way by the patient. The words spoken by persons with syntactical aphasia are expressions of the speech functions which are not disturbed. Head disagreed with Collier strongly in his conceptions of visual speech or auditory speech; he says that when a person speaks "his actions are carried out by psychological processes above the visual and auditory levels." Head explains that his object in selecting the cases studied by him was to see the actual forms assumed by destruction of speech regardless of the nature of the lesion, whether it was cortical or subcortical . . . "it was of no use trying to localize the position of an unknown function on the surface of the brain." He strongly objects to the use of the term "faculty" as applied to speech and intelligence and says that such conceptions come down from medieval philosophy. He is particularly strong in his objections to the rather old and conservative stand taken by Dr. Stewart.

What Head thinks about Wilson's discussion is summed up in his statement that "the moment one drove apraxia to definition one saw that it did not exist . . . but when one used the word apraxia to explain the loss of speech it did not seem to explain more than the most simple of aphasias. He wished to get rid of all such terms."

In answering Dr. Barnes, Head said that he did not believe in "localization . . . of so-called motor aphasia or sensory aphasia" because he did not believe in the existence of motor and sensory aphasia." Head agrees with Jackson that there is no such thing as general memory, and that psychologists nowadays did not believe in general intellectual defects. There are only definite things remembered. There is no such thing as general memory. "Speech is a form of intellectual activity; therefore, in saying there was a defect of those processes which were responsible for speech, it was assumed there was a defect of intellect. But this was a specific loss of function and there was not that gross widespread defect of intellectual aptitudes as was found in the slightest case of dementia."

To this point the reviewer has called attention previously. He is in accord with Head, the only difference being in the interpretation of what intellect and intelligence mean. If one does not use intelligence as a general term, meaning by it that he is intelligent who has attended to and stored correctly many memories and clearly conceives their importance and significance, then he may come into complete agreement with Head. For Head and many psychologists there is no such thing as general intelligence but merely special intelligences or memories of specific things. This the reviewer, from the medical clinical standpoint, thinks is mere splitting of hairs.

Osnato, New York.

DEVELOPMENT OF THE REACTIONS AND PLANTAR REFLEXES OF A BABY BORN BEFORE TERM AND UNTIL TWO YEARS OF AGE. H. Bersot, Schweiz. Arch. f. Neurol. u. Psychiat. **7**:213-231, 1920.

The researches of Flechsig, Soltmann, and others, relating to the development of the anatomic structures of the nervous system placed our knowledge of this far in advance of that concerning the development of its physiologic functions. Structurally the nervous system develops rapidly during the first years of life, progressively less rapidly up to the twentieth year, after which involution slowly commences. Concerning the physiologic development much less is known, particularly the development that occurs during the intra-uterine period.

In attempting to establish the relationship between the anatomic features of the central nervous system and its function, the conclusion was reached that one must naturally have preceded the other. Most writers state that full development of given parts of the organ precede the development of their function, while others believe that function may precede the full development. An indeterminable discussion has resulted, centered mainly around these facts: Up to the middle of the fifth month only gray matter is found in the fetal nervous system, after which myelinated fibers appear; on this fact the assumption was based that the first movements of the fetus are noted in the fifth month of intra-uterine life. In contradistinction to this, Preyer states locomotor movements are noted from the seventh week of embryonic development. A similar dispute has centered about the observation that after the sixth month following birth there is a rapid disappearance of the extension of the large toe on plantar irritation, which is coincident with the development of myelinization of the pyramidal tracts; that incomplete myelinization of the pyramidal tracts is the cause of the Babinski reflex.

What characterizes the responses to external excitation obtained in an infant is the fact that they are generalized. The organism as a whole responds. The baby cannot localize, cannot vary its reactions and does not differentiate. In this respect it differs fundamentally from the adult, in whom a particular excitation produces a localized response that varies as the excitation varies. Thus, in a baby, in tapping the back with a pin, motor reactions appear in both lower extremities and toes. In the adult, on similar excitation, one obtains only a feeble local reaction, the lower members remaining immobile. If the excitation is more marked and painful, one obtains, in an adult, reactions which approach those in a baby following slight excitation. The reactions which are not produced in the adult on slight excitation are supposed to exist in a latent manner and are not completely abolished. Therefore, there is no fundamental difference in the reaction of a baby as compared with that of the adult; the difference is one of degree, variability and multiformity.

It is generally stated that during the first months of life the Babinski reflex turns out positive. C. Cattaneo found it present in 30 per cent. of cases during the first three months of life, then progressively less frequently up to the end of the second year. According to Muggio, after the sixth month of life, it is seen especially in rickets. According to Lowest-Morse, the normal plantar reflex is not seen until after the second year. Léri, from a study of 166 infants, concluded that at birth extension of the toes is the rule, flexion is the exception. After 3 years, the opposite is true. Between the ages of 1 and 3 extension of the toes is rather indicative of affections of the pyramidal tract. The pyramidal tract, having scarcely had its myelinization completed, is more sensitive to toxic degenerations than that of the adult and is readily affected in general nutritional disorders. According to him, extension of the toes normally disappears at about the fifth or sixth month. This disappearance is coincident with the disappearance of the general spasmodic attitude of the new-born.

S. Galant, in studying the plantar reflex of 132 infants, found the sign of Babinski present in ninety-eight cases and fanning of the toes in thirty-five; the latter is seen especially in babies up to the fourteenth day; after the second week fanning of the toes is rare. Galant noted that the older the child, the less frequent the Babinski phenomenon and the more restricted the reflexogenic zone. By repeated excitations of the sole, except in very young babies, one is able to suppress the Babinski sign and elicit the so-called normal plantar reflex.

The reflexes of a baby cannot be considered as fixed and immutable as those of the adult. It is this variability that Bersot is particularly interested in. In preceding studies of reflexes during the entire life, Bersot showed that the plantar response diminished in intensity until the twentieth or thirtieth year, at which time it reached its most feeble value, and following which it again became augmented. He showed that a plantar reflex was present during intra-uterine life, when it took the form of part of a general reaction, becoming more and more differentiated and localized after the third or fourth months following birth. Fortunately, Bersot was able to obtain a fetus to carry out his study.

According to Preyer, movements appear after the seventh week; these motor reactions have characteristic features. Soltmann states that the excitability of the fetus is less feeble; that the muscular movement possesses a certain slowness of reaction such as is seen in the adult on great fatigue or in a number of pathologic conditions. According to A. Westphal, fetal muscles are inexcitable by the electric current. Movements interpreted by Ahlfeld and Reubold as deglutition and by Mermann and Ahlfeld later as regurgitation are also seen. Ahlfeld also stated that rhythmic movements at the rate of 50 to 60 times per minute are visible to the attentive observer and correspond to movements of the thoracic wall of the infant. These have been traced by kymographic methods and have been interpreted as respiratory movements which are so slight in amplitude that the amniotic fluid does not enter the lungs. The fetus studied by Bersot was of four to five months', seventeen to eighteen weeks' development; it behaved very much as in a state of sleep in which external excitation could scarcely arouse it. On irritation, feeble movements were noted in the extremities, the fingers and toes were immobile, the respiratory movements were slight, although the mouth was opened and there was no cry. As soon as stimulation was discontinued the infant at once seemed to plunge into a deep sleep.

Plantar excitation in this premature infant provoked only a flexion of the toes which was slow and feeble, but definitely present, and included all of the toes, particularly the outer four. The great toe frequently remained immobile. Of the four outer toes, the movement consisted principally in flexion of the first phalanges and extension of the two others; the toes seemed to lengthen and at the same time to go through a movement of slight plantar flexion, such as that produced by the combined action of the lumbricales and interosseus muscles. This movement was not accompanied by any other movement of the lower extremity or in the rest of the body and ceased after the sixth to eighth stimulation as though rapidly fatigued. This type of reaction was also noted by K. Crabbe. Oppenheim's reflex was not obtained.

At twenty-seven to twenty-eight weeks of intra-uterine life the infant presented the same reactions as those noted at birth. Irritation was accompanied by feeble crying of short duration. Plantar irritation here produced a retraction of the entire lower extremity by flexion of the thigh and leg which ceased as soon as the irritation was removed. The toes also went through a movement of flexion and elongation. The large toe remained either immobile or followed the movement of the other toes. There was also a contraction of the tensor fascia lata. On the opposite side the toes went through the same movement of extension with slight dorsal flexion of the foot.

At thirty-four to thirty-five weeks of fetal age the external appearance was similar to that of an infant born at term, except for a looseness of the skin due to lack of subcutaneous fat. The responses to irritation are more or less universal and are spasmodic or athetoid in character. On plantar irritation the four external toes undergo an extension, which is sometimes noted in the great toe also; however, more often this toe extends as in a positive Babinski reflex. The extremity is flexed at the ankle, the knee and the hip. Contractions of other muscles, such as the quadriceps and tensor fascia lata, are noted. The opposite extremity goes into a position of extension throughout. It is noteworthy that a baby born at thirty-five weeks has not the same reflex activity as that thirty-five weeks of age, but born at the thirtieth week. In the latter the reactions are more extended, more numerous and less feeble, as though reflexitivity developed by external irritation.

The reflexes of an infant born at term and up to six months of age are very much like those just described. The motor reactions are a little more lively, stronger and extended. Excitation of the foot usually produces a flexion of the toes rather than a positive Babinski reflex; however, extension of the large toe appears in the contralateral extremity before it appears in the homolateral one. At two or three months plantar excitation no longer produces flexion of the great toe, but a strong extension of the large toe and a movement of dorsiflexion of the foot. In the other toes there is an intra-osseus and lumbricales reaction. Contralaterally, extension of the great toe occurs with force and slowly and often remains at hyperextension during the entire observation of twenty excitations, which it is the custom of the writer to produce. There is less tendency to fatigue, and the feet tend to retain their position of defense for some time after the excitation has ceased. The general reaction, however, is marked.

At five or six months of age the reactions on the whole are the same as at the third month. These reactions constitute the plantar reflex. There is absolutely no way of distinguishing them as they occur normally from those noted in pathologic conditions at the same age. Bersot agrees with most writers that the Babinski phenomenon is present in the majority of cases. An important point, he thinks, is the generally increased reflexitivity of the infant. The entire cutaneous covering is reflexogenic, particularly up to the second or third month.

After three or four months the reflexogenic zones commence to become more limited. The plantar reflex can be elicited only by excitation of the plantar surface of the foot. In infants of six months the reactions are analogous to those of three months.

At nine to twelve months a distinction arises in that the infant seems able to discern different types of excitation. It does not react in a uniform manner to all external excitations. It remains quiet when it is uncovered, movements become better coordinated, and a life of relation seems to establish itself. Plantar irritation leads to retraction of the excited member with extension of the contralateral one. The toes usually go through a movement of plantar flexion, in which the large toe participates. There may be a flexion of both phalanges or flexion of the first with extension of the others. Extension of the contralateral great toe has completely disappeared by the age of nine to twelve months. At times it appears on the homolateral side, but more often has become replaced by flexion. The reflexogenic zone has become very much restricted.

At eighteen months, extension of the great toe is rarely observed. The general reaction of the organism is much diminished, muscular contractions are more rapid, movements quicker and more forcible, and the reactions themselves diminished in number, intensity and duration. Bersot's study led him to believe that the reaction in infants in particular must include all of the reflex movements rather than a limited observation of the particular member irritated. The article will be continued.

Woltman, Rochester, Minn.

CONCERNING THE DIAGNOSIS AND TREATMENT OF HYPOTHYROIDISM. N. W. Janney and M. E. Henderson, Arch. Int. Med. **26**:297 (Sept.) 1920.

The authors emphasize the fact that early cases of hypothyroidism are frequently overlooked, although frank cases are easy to recognize. They report a series of cases in which, besides the ordinary history and physical examination, differential blood counts, glucose tolerance tests and basal metabolism determinations were made. The technic for the glucose tolerance test consisted in giving 1.5 gm. glucose per kilogram of body weight in 3 c.c. of water per gram of sugar and samples of blood taken immediately before the ingestion and at hourly intervals for two or more hours thereafter. Basal metabolism determinations were made with the Benedict portable apparatus.

Seventeen cases are included in the report, one of which was a definite cachexia strumipriva, and four were classed as dysthyroidism. As a group, the most important and frequent findings are: history of obesity, particularly in early life; history of glandular disturbances in the family; mental symptoms embracing dulness, irritability, headache, melancholia, poor memory and exhaustion; anomalies of development and liability to infections; physical findings which embraced hair anomalies, dry, harsh skin, pigmentation, atrophy, frail, poorly developed nails, cold extremities and skin, stubbiness of hands and feet, dull expression, heavy lips, large tongue, saddlenose, increased panniculus, fat pads on the dorsum of hands and feet, in the supraclavicular fossae and on the back; subnormal temperature, pulse and respiration. The thyroid gland varied in size from normal to enlarged or decreased. The blood picture usually showed a mononucleosis. The average percentage in thirteen cases was: polymorphonuclears 55.1 per cent., lymphocytes 36.1 per cent. and large mononuclears 3.53 per cent., eosinophils being present in only one case.

Basal metabolism determination is pointed out as being the best means of establishing direct evidence of thyroidal disturbance, but it cannot be used as a sole and absolute criterion. The authors state that the glucose tolerance estimation showed that a hypoglycemia and a lowered blood sugar curve existed in hypothyroidism much more commonly than in hyperthyroidal conditions. In relation to the blood sugar curve it is pointed out that normally there is a return to normal in one and one-half to two hours, but in hypothyroidism it is considerably delayed and indicates a faulty sugar metabolism in the system. It must be remembered, however, that the same picture is sometimes given in disorders of the endocrine glands other than the thyroid. The basal metabolism rate did not follow the sugar curve in any sense, and it is concluded that the sugar curve is more reliable as diagnostic evidence earlier in the disease than basal metabolism ratings. The therapeutic test for deficiency of thyroid secretion is mentioned as filling a proper place in the diagnostic armamentarium and not to be forgotten.

Treatment is best controlled by basal metabolism studies since it indicates overfeeding or underfeeding earlier than the clinical evidence, although it must not be forgotten that in older persons it may be misleading, as the ability to respond is much slower clinically than in younger people, and a basal metabolism rate may be read as normal while the patient shows no change. So far as preparations of the thyroid gland are concerned in therapy, the authors emphasize the importance, scientifically and physiologically, of thyroxin which produces a much better response than any other preparation.

In regard to nitrogen metabolism in a person with hypothyroidism, one of the authors has shown by experimental work (reported elsewhere) that there is difficulty of assimilation of digestive products from the gastro-intestinal tract, and further that after the assimilation there is also difficulty with the utilization of the materials, due to thyroidal inactivity. In two cases of this series nitrogen balance was determined, the patients showing definite and prompt recovery after proper treatment, to the extent that food was better assimilated and utilized, as evidenced by the beginning of growth and the laboratory data.

The article further deals with functions of the thyroid gland, and in summary form the authors detail certain conclusions. The picture of hypothyroidism is one of arrested development, degenerative processes of the tissues, lessened food absorption from the alimentary tract, poor utilization of the absorbed digestive products, abnormal carbohydrate metabolism, decreased excretion of purin bodies and the appearance of urinary creatin and general lessening of all metabolic activity—all caused by a hormone hunger due to insufficient thyroid secretion.

Patten, Philadelphia.

STEINACH'S OPERATION. G. Hotz, Schweiz. Arch. f. Neurol. u. Psychiat. **7**:344-349, 1920.

In 1910, Steinach advanced experimental evidence to show that rats in which the male gonads had been removed and replaced by female gonads took on feminine attributes, and that the reverse occurred when ovaries were replaced by testicles. This applied to both physical and psychic characteristics, which are probably controlled by the cells of Leydig in the testicle and the luetin cells in the ovary. Bouin and Ancel, in 1903, showed that ligation of the vas deferens interrupted spermatogenesis, but led to hypertrophy and hyperplasia of the interstitial cells. Tandler and Gross, in 1908, produced the same effect by roentgen-ray radiation. These animals retained their secondary sexual characteristics and libido.

With this as a basis, Steinach attempted to bring about a regeneration of the involuting gonads by ligaturing the vasa deferentia, later by passing a ligature between the testicle and the epididymis. The aging rats were "rejuvenated" in every respect and sexual activity restored to an unexpected degree. The life of these animals was reported to have been lengthened by seven months, which is about one-fourth. The second senium advanced uninterruptedly, however.

The operation was carried out on man by Lichtenstern by transplanting the testicles of cyptorchids. Steinach's designation of "rejuvenation," *Verjuengung,* precipitated a great deal of comment on the part of the general press.

Hotz was very much interested in this work and reports a case of a 50-year old merchant, who presented himself with the request that this operation be performed on him so that he would be able to carry out his business responsibilities more efficiently. He had noted that his memory and grasp of the business situation had diminished during the past four years, and felt that it would soon be necessary for him to give up his work. The patient was of the nonsmoking, totally abstinent, vegetarian, homeopathic and nature-healing type. The operation was carried out as he requested, no mention being made to him of the questionable value of such a procedure. Six weeks later the operation had its full effect; the patient stated that he could work as quickly as he could ten years previously, that his memory was exceptionally good and his sexual powers increased. On the latter point, however, he laid little stress.

The experience of a younger man is recounted, who had had the operation performed because of failing libido, with absolutely no benefit.

Hotz performed the experiment of transplanting ovaries and testicles into the belly-wall of rats with the view of determining the viability of transplants. Without exception, he found acute necrosis of all testicular tissue; the cells of Leydig, however, persisted somewhat longer, but all of them were necrotic after a period of four weeks. The fate of the ovaries was similar. In previous work he had transplanted the thyroid in cretins with the result that in five months the entire tissue had been organized into a fibrous scar.

Experiments on rats cannot be looked on as the full equivalent of a similar condition in man, as the testicles of a rat are much larger in relation to body-weight, the amount of interstitial tissue comparatively greater and sexual activity more marked. As a matter of fact, the operation of tying the vas in man is not new and has been done for about twenty-five years in order to bring about a reduction in prostatic hypertrophy; the results have never suggested a rejuvenation.

The writer also called attention to the fact that in epididymitis, arteriosclerosis, diabetes, carcinoma, alcoholism, cirrhosis of the liver and other conditions in which atrophy of the spermatogenic tissue occurs, there is an increase of the interstitial tissue without any suggeston of increased vitality.

Tandler and Gross have shown that in moles there is a decrease of interstitial tissue coincident with increased spermatogenesis during the mating season, and an increase during the period of diminished spermatogenesis.

The conclusion seemed apparent to Hotz, therefore, that atrophy of the spermatogenic tissue is primary. Lessening of tissue pressure follows, which in turn leads to a compensatory hyperplasia of the interstitial tissue. He also had the opportunity to observe a patient in whom an epididymectomy was performed on account of tuberculosis on the left side five months previous to a similar operation on the right; a careful study of the testicles showed absolutely no difference on the two sides.

The writer feels, therefore, that he cannot indorse the views of Steinach and Lichtenstern. He is convinced that Steinach's operation is valuable only as a powerful suggestion.

WOLTMAN, Rochester, Minn.

A CASE OF ENCEPHALITIS LETHARGICA INVOLVING CHIEFLY THE CEREBRAL CORTEX. G. A. WATSON, Neurol. & Psychopath. **1:**1 (May) 1920.

The case reported is that of an anemic woman, aged 28. Clinically she presented a picture of agitated melancholia, having attempted suicide by cutting her throat a few days before admission, and after admission by trying to tear the wound open. She remained in this mental condition without change for four months. Then for three days she seemed to be very ill and vomited several times. During the ensuing two months she seemed to improve mentally, but, at the end of this time, began to complain of pain in the back and down the left thigh, and she limped when walking. She was confined to bed. This condition continued for two months, then she lost her power of speech and the right side of the tongue became paralyzed (eight months after admission). She was drowsy, disinterested and "lethargic," but could be aroused to answer questions. Eleven days later there was a right hemiplegia involving the face, arm and leg, with anesthesia and analgesia including the right half of the tongue. The tendon reflexes were especially brisk on the right, although the right plantar and abdominal reflexes were absent. Optic neuritis of moderate degree was present. A week later marked atrophy of the right arm, forearm and hand and of the left calf and thigh were noted. She died the following day. During the illness her temperature was subnormal, and the blood count showed a definite leukocytosis.

At necropsy the brain was found to be soft, and in the left hemisphere there was a large area of recent softening with adherent membranes. After hardening, the sectioning of the area disclosed streaks of vascular congestion that was not grossly apparent in the rest of the brain, but no hemorrhage. Microscopically the membranes showed increase of vessels with marked congestion and here and there small hemorrhages into and beneath the membranes. In the area of softening there was an enormous increase of vessels with marked cellular proliferation, mainly around the vessels, but in the most affected parts spreading widely into the surrounding tissue. The proliferative elements were lymphocytoid, plasma and pseudoplasma cells, but mainly polyblasts, epithelioid and reticulate types of cells, obviously originating from the adventitia. Neuroglia cells were somewhat increased, and the endothelium of the vessels showed proliferation. Small hemorrhages were found around the vessels, though they were not numerous except in the putamen and precentral convolution, where they were rather large. Many veins showed partial occlusion by thrombi. Around many vessels were rarefied sievelike spaces—appearing like an exudate—especially in the white matter, and containing amorphous deposits and proliferated cells which stained well. Nerve cells were affected in the softened area corresponding to the patchy intensity of softening. The precentral and subfrontal regions, the insula, putamen and part of the temporal lobe were mostly involved. The putamen seemed to have been most affected showing intense vascular and cellular increase with destruction of cells, hemorrhages, perivascular sievelike areas and chromatolysis, the remaining portions of the basal ganglions being little involved. In the temporal region the process appeared to be of longer standing and not as acute. In other parts of the brain there was some increase in vascularity, and the vessels showed some adventitial proliferation, increase of glia cells in the vessel vicinity and general cell destruction—although nowhere was the process as marked as in the softened left hemisphere. The medulla and pons showed thickened membranes which were infiltrated, the vessels congested and containing hyalin thrombi; occasionally there was a small hemorrhage. There were adventitial proliferation and glia cell increase and much infiltration with round cells, most marked in the medulla. The cord showed similar changes of the membrane and vessel and small, round-cell infiltration in the gray and white matter. The nerve roots and ganglions evidenced congestion, cellular proliferation and in the ganglions small hemorrhages.

The condition is termed a meningo-encephalo-myelitis on account of its widespread distribution, but the chief stress occurred in the left hemisphere in the areas mentioned.

Patten, Philadelphia.

STATISTICS OF EPIDEMIC ENCEPHALITIS. I. S. Wechsler, Neurol. Bull. **3**:87 (March) 1921.

Wechsler obtained accurate information concerning 864 cases of epidemic encephalitis, from private as well as hospital records. Although the entire country had fair representation, more than half of the cases were gathered from New York City.

The age incidence ranged from 4, 6 and 7 weeks to 84 years, the greatest number of cases occurring between the ages of 20 and 50. The period of childhood and adolescence was much less affected than it was in the recent poliomyelitis epidemic. Males were attacked much more severely than females, the proportion being three to two. Practically every occupation was represented. Seventeen physicians, almost 2 per cent. of the group, were included. Fifty-four per cent. of the patients were foreign born. As most of the cases occurred in adults over 25 years of age, the majority of the patients were married. Pregnancy was a complication in twenty-two cases reported. Four of these went to a fatal termination. Wechsler emphasizes the low mortality in this type of disease compared with that of influenza when complicated by pregnancy.

Regarding familial incidence: In five instances the disease occurred in two members of a family. In view of the practical absence of contagion in hospital cases (although two interns were among those reported) direct transmission is considered a negligible factor.

Wechsler found a fairly low mortality rate. Of 850 patients, 178 died, a percentage of almost 21. Death occurred chiefly in those presenting psychotic, delirious and meningitic symptoms with high fever and acute onset.

The majority recovered more or less completely, but many manifested sequelae, especially those of the parkinsonian type, and those having ocular disturbances, tremors, abnormal involuntary movements, etc.

Under the heading of clinical forms and anatomic localization, over twenty types of the disease are included. The lethargic group leads with a percentage of 35. The parkinsonian type, assuming second place, was present in 15 per cent. of the cases.

McKendree, New York.

A CONTRIBUTION TO THE STUDY OF GLIOMA. S. Uyematsu, J. Nerv. & Ment. Dis. **53**:2 (Feb.) 1921.

This important article opens with a discussion of three basic questions: (1) the differentiation of glioma from sarcoma; (2) the pathogenesis of glioma, and (3) the relation between pathologic proliferation of neuroglia and tumors of neuroglia origin. The author has studied four brains showing gliomatous growths, not only as regards the tumor itself but also the general glial condition in the entire brain.

The first case studied was one diagnosed clinically as paresis. At necropsy a central glioma of the third ventricle was found which on microscopic section proved to be a glioma, and throughout various areas of the brain there was a general increase of glia cells, without perivascular infiltration and no disturbance of the cyto-architecture and myelo-architecture, and no changes in the nerve cells. The changes in the neuroglia consisted not only of increase in the cells but also of a deformation of the normal spheric type of nuclei. These neuroglia changes were most marked in the left cornu ammonis, the right postcentral convolution and both frontal lobes. Histologically no distinction was noted between the tumor and the gliomatous areas. The cornu ammonis did not show increase in volume, making it difficult to classify the process. According to the histology which was identical with the tumor itself, it ought to be called a diffuse glioma or diffuse gliomatosis, but when the gross picture was taken into account it seemed to the author more justifiable to classify it as a diffuse sclerosis.

The second case was also diagnosed clinically as paresis and at necropsy showed a superficial glioma of the left frontal lobe. On microscopic section no evidence of paresis was found. The tumor itself consisted of glia cells and fibers. The cells showed two different types: one small, like the normal glia cells; the other larger, appearing more like ganglion cells of the cortex or the anterior horn of the cord, which showed the characteristics of the granule cells of Merzbacher. A tumor of this nature was designated by Stroebe as glioma gangliocellular. The author suggests the name granule cell glioma or giant cell glioma. This case also showed a marked general gliosis, not only in the cerebrum but also in the cerebellum, pons, peduncles, medulla and cord; not of the same intensity throughout but showing considerable local differences. The pons and the occipital pole of the right hemisphere showed the most marked gliosis. In this case there was found in the white substance of the right occipital lobe a small cavity lined with cubic epithelial cells in places in a single layer, in others 4 or 5 cell layers deep. It was not connected with the lateral ventricle, but in general appearance it was exactly similar and was considered to be a displaced or invaded neural canal.

The third case was that of a superficial glioma of the left temporal lobe; microscopic section showed it to consist of many large cells like those in the preceding case and a smaller number of small spheric cells, like the normal neuroglia cells of the white matter. Through the brain a general increase of glia was noted, but differing from the first two cases in that the nuclei did not vary so much in shape, being more or less spheric.

The fourth patient whose case was diagnosed clinically as traumatic psychosis had a tumor of the left internal capsule that consisted of more cells than fibers, especially in the center. Where the neuroglia cells were closely packed, epithelial formations of various forms and sizes were encountered, which on higher magnification were seen to show the characteristics of ependymal cells. The neuroglia cells through the brain were increased; the nuclei were more or less altered, normal, spheric cells being rarely observed.

In the summary the author discusses the question of the diffuse gliosis which he found in all of his cases of glioma and absent in other tumor cases, used as control, which were not glioma. The theory of Storch that some stimulus cast forth from the tumor is the proliferative stimulus is not accepted by the author because of the distant gliosis. The second theory of intracranial pressure producing it was ruled out by the absence of gliosis in other tumors. The third theory, that of Ranke, that tumor cells may wander out from the tumor into neighboring tissue is also insufficient. The author's opinion is the assumption of a common causative factor for both general gliosis and tumor formation, such as happens in diffuse sclerosis.

The article is well written, the pictures are good, and an immense amount of work has been done. The general increase in glia throughout the entire brain in glioma, while not new with this author, is brought out forcibly by him.

Winkelman, Philadelphia.

ON THE SYMPTOM COMPLEXES OF LETHARGIC ENCEPHALITIS WITH SPECIAL REFERENCE TO INVOLUNTARY MUSCULAR CONTRACTIONS. F. M. R. Walshe, Brain **43**:197-219 (Nov.) 1920.

The author discusses the remarkable clinical diversity of lethargic encephalitis and suggests that the numerous schemes of classification and description adopted, although inevitable in the early and growing stages of knowledge of so polymorphic a disease, tend to confuse rather than to lend precision to our conception of this disease, for practically every case presents simultaneously, or at some phase of its course, the features of several clinical types. As an example of the ineptitude of purely symptomatic classification, he briefly discusses myoclonic encephalitis.

He suggests that the use of focal nervous signs for the purpose of defining types would insure a uniform terminology in the description of the disease and so obviate much of the confusion in the literature. On this basis, the author contrasts acute poliomyelitis and lethargic encephalitis. He further illustrates the points discussed by seven personally observed cases showing various positive motor symptoms. He draws the following conclusions: 1. "It seems probable that the psycho-motor excitement of the 'choreiform' manifestations of lethargic encephalitis, the jacksonian fits, and the rhythmic fibrillary, the fascicular and muscular contractions collectively grouped by various writers as 'myoclonic' symptoms are in fact the expressions of an irritative or exciting action of the virus of the disease on neurons of the three physiological levels (Hughlings Jackson) of the nervous system, and that such symptoms can be localized on the same principles as are employed to localize negative or paralytic symptoms.

2. "If we accept this view of the irritative origin of the motor symptoms under discussion, a striking feature emerges; namely, that where the virus

produces negative or paralytic symptoms it has, ever since the first appearance of the disease, shown a definite and characteristic selective action on the cells of the basal ganglia and on those of certain motor nerves in the brain stem, producing the familiar basal ganglia and midbrain types of the disease with its associated lethargy.

3. "In its irritative or exciting effect the virus appears to act equally on any and every part of the nervous system, from cerebral hemispheres to spinal roots, hence the polymorphic character of cases showing what we may call irritative symptoms. It is such cases that present the many difficulties in any attempt to classify lethargic encephalitis into clinical types. Possibly we may be dealing with a complex virus in which more than one active component exists."

Blitzsten, Chicago.

PSYCHOSIS ASSOCIATED WITH TETANY. A. M. Barrett, Am. J. Insan. **76**:373 (April) 1920.

A psychosis associated with tetany has rarely been observed. Barrett reports two such cases and gives a good summary of the literature on the subject. In the first case a mental condition preceded the tetany, while the reverse order obtained in the second.

In Case 1 the psychosis followed a domestic sorrow and was characterized by depression, loss of weight and sleep, indicanuria (as high as 100 mg. in twenty-four hours), achlorhydria, periods of stupor, retardation and irritability. Soon after, neurologic symptoms appeared, manifested by spasticity, tremor, increased reflexes, flexion of the elbows and adduction of the thumbs —these positions being more or less constantly maintained. The movements were ataxic. The patient was constipated. The mental unclearness progressed with apprehension, disorientation, irrelevancy, confusion and hallucinosis. There were jerking and twitching movements of the upper half of the body— often brought out quickly by merely tapping any of the muscles. Chvostek and Trousseau phenomena were present. Ten weeks after admission the stupor deepened, and the twitchings became marked. Several days later he complained of pain in the back of his head and arms, and the characteristic accoucheur position was assumed. He then became stuporous and restless by turns, and the tetanoid contractures came on at intervals evidently accompanied by considerable pain. Nearly five months after admission he died in a condition of considerable weakness, but with persistence of the muscular irritability. The necropsy diagnosis was "ascending thrombo-phlebitis of the right iliac and femoral veins; embolism and thrombosis of the pulmonary vessels; purulent pneumonia; emphysema; atrophy and passive congestion of all organs." In the nervous system a large cyst in the anterior lobe of the pituitary gland near the base of the stalk was discovered. The parathyroid glands showed no abnormality on histologic examination. The nerve cells in the cortex showed marked chromatolytic changes and fatty pigmentation.

The second case began with a sudden attack of sharp pains beneath the right patella. Soon the leg dragged and became stiff. The stiffness extended into the thigh and up the back. Bending on arising from a sitting to an erect posture was difficult. The condition grew progressively worse until, two years before his admission, his eyesight began to fail and he fell frequently, lying with all parts of the body in marked flexion. A year later he became mentally unclear, had somatic delusions, was depressed and untidy, confused at times, disoriented, irrelevant and often quite incoherent. Neurologically an increase and disproportion in reflexes occurred, tremors developed, the tongue deviated to the right and the ocular fundi showed venous congestion. After three weeks he remained mentally clear and was discharged. The muscular irritability continued, however, and was characterized by tonic contractures, tremors and jerking movements. He was readmitted three months after discharge. Except for memory defect, his mind was clear. Neurologic examination at this time revealed increased reflexes in the upper extremities, the Babinski reflex bilaterally, speech defect, sluggish pupils, weak and staggering gait, loss of sense of position in the toes and spontaneous jerkings of the arms and legs at intervals. He then became stuporous. Generalized tenseness and contraction of muscles developed with tremors at irregular times. The Chvostek and Trousseau phenomena were present. The position of the hands and arms was typically accoucheur during the attacks. Death occurred nine days after the second admission. Postmortem findings failed to reveal changes in the parathyroid glands, but the nerve cells in the cortex showed widespread and severe alterations. The neuroglia and blood vessels also shared in the degenerative processes. The areas affected included practically all parts of the central nervous system. The pituitary gland showed many small cysts in the posterior lobe and an exceptionally large one near the base of the stalk.

The author suggests the original disease in the pituitary glands as the causative factor in the altered metabolism producing secondarily the disorder of the nervous system and the dysfunction of the parathyroid glands.

Patten, Philadelphia.

PIGMENTARY RETINITIS AND HYPOPHYSIAL OBESITY. Chaillous, Ann. d'Oculistique **158**:100.

The common ocular involvement in disease of the hypophysis is bitemporal hemianopsia. Retinitis pigmentosa, with obesity and polydactylism as a symptom triad, was described by Bardet in his theses of 1920.

The author reports the case of a patient, a girl aged 4 years, who consulted him in 1914, because the parents said her vision was affected. Enormous obesity of the patient was striking, but the eyegrounds were negative.

In 1919, this patient was again seen, and the obesity was even more marked especially of the abdomen and legs, but no points of tenderness were present. The ocular symptoms were increased. Vision was so reduced that she had to grope her way about. Examination of her eyes showed a slight external strabismus but no nystagmus. The pupils were normal. The disks were slightly discolored on the temporal side. At the periphery of the fundus were small patches of choreoretinitis, such as those seen in pigmentary degeneration of the retina. A radiograph of the skull showed the sella turcica to be normal, while Wassermann reactions of both the child and the mother were negative.

Thus, in this patient were found obesity, hexadactylism and pigmentary degeneration of the retina. Pituitary therapy was tried with no success.

A discussion of Bardet's paper follows, which tries to show a relationship between pituitary disease and retinitis pigmentosa. Chaillous cannot agree with his reasoning, from the evidence produced, although he thinks a new field has been opened up in the study of such conditions, in which the radiographer, neurologist and ophthalmologist may well cooperate.

Scarlett, Philadelphia.

THE TREATMENT OF CEREBROSPINAL FEVER. C. Worster-Drought, Neurol. Psychopath. **1**:1 (May) 1920.

Early diagnosis and treatment are of paramount value in this disease. It is not harmful to inject serum when on lumbar puncture the fluid is found turbid. In the premeningitic stage, especially if there are suspicious petechial spots and premonitory signs, intravenous administration of serum is a wise procedure, as it has been conclusively shown that infection occurs through the nasopharynx via the blood stream. Serum can be administered intravenously with safety up to 200 c.c. or more. Early examination of the spinal fluid may disclose the presence of the organism before there is much cellular reaction and before the fluid becomes turbid, hence the advantage of examining smears from centrifugalized fluid. The intravenous administration of serum after meningitis has developed is of doubtful value due to the loss of permeability of the membrane of the subarachnoid spaces. At this stage, however, intraspinal administration is of great advantage since it concentrates the antibodies and immune principles where the bulk of the disease process is found. Furthermore, it has been shown that absorption into the blood takes place quite rapidly from the subarachnoid spaces, and it is best to repeat the injections at least every twenty-four hours. The author uses as a total from 120 to 600 c.c. of serum in his cases, employing the gravitation method of administration since it is much less likely to produce respiratory failure than the syringe method. He advocates the continuance of the injections until decided improvement has taken place clinically and the fluid is free from organisms. After the discontinuance of the serum he continues to drain the intrathecal space, believing it assists in carrying away waste products and increases the inflow of healthy fluid. At the first sign of recrudescence he repeats the serum injections without delay. Vaccines are given; 250 million organisms are injected subcutaneously during the first three days of the illness and the number is increased by 500 million at each succeeding injection (every fourth day) up to a maximum dose of 2,500 million. Polyvalent vaccines are best while waiting for the determination of the type of meningococcus and the preparation of autogenous vaccines. The polyvalent vaccine represents strains of all four types based on the Gordon classification.

As far as general management and treatment are concerned, besides the usual things, the author advocates keeping the patient's head low, changing from a liquid to a semisolid diet as soon as possible, administering phenacetin and caffein for headaches, acetyl-salicylic acid for the muscular pains and hypnotics for sleeplessness. Reinfection through the nasopharynx is prevented by swabbing with chloramin-T (1 per cent.) as soon as possible after the onset of the disease.

Of seventy-two patients treated by the author as outlined, fifty-seven recovered and fourteen died. Thirteen of the fatal cases were of the fulminating type, acute fatal, or progressively purulent types. Of the fourteen who died only one had internal hydrocephalus. having been admitted three days after a recrudescence set in. The average duration of illness of those who recovered was fourteen days, and only two or three gave evidence of symptoms of internal hydrocephalus during the disease.

Patten, Philadelphia.

THE POSTENCEPHALITIC PARKINSONIAN SYNDROME. J. A. Barre and L. Reys, Bull. méd. **35**:351 (April 27 and 30) 1921.

The parkinsonian syndrome has been relatively frequent in the course of epidemic encephalitis. In a series of 101 cases which the writers have studied, it developed in twenty. They emphasized that, contrary to other reports, the parkinsonian syndrome sometimes accompanies or follows a myoclonic encephalitis.

They review first the symptoms which are common to Parkinson's disease (paralysis agitans) and to the parkinsonian syndrome (referred to as the pseudoparkinsonian syndrome) which develops in certain cases of epidemic encephalitis.

In the pseudoparkinsonian syndrome the muscular rigidity has a predilection for, and reaches its maximum in, a cervicofacial distribution. Likewise, tremor in the pseudoparkinsonian syndrome is most often seen in the face.

Slowness of voluntary movements and absence of spontaneous movements is present in both. Although this slowness of movement almost always is accompanied by rigidity and tremor, it has been observed apart from these.

Disturbances of equilibrium, vertigoes and nystagmus occur in both. They are moderately rare in true Parkinson's disease and much more frequent in the postencephalitic syndrome. The acuteness of the developing infectious lesions explains this prominence in the latter. The oculomotor signs, so outstanding in the pseudoparkinsonian syndrome, are not totally unknown in the true. The vasomotor disturbances appear, to the writers, to be the only features peculiar to Parkinson's disease.

The encephalitic parkinsonian syndrome never appears insidiously or without having been preceded by other pathologic manifestations generally of an infectious order. With this point in mind, it is possible to go further and distinguish three types of onset of the syndrome. 1. It constitutes an initial manifestation and dominates the encephalitis. 2. It develops as an immediate sequel of the encephalitis when the other manifestations of the latter still are evolving. 3. It appears during the convalescence.

The concluding portion of the paper deals with therapy. The writers recommend scopolamin and atropin used in alternation. The latter is used in a dose of 1 cg., given two times a day. Atropin administered in this way can arrest the evolution of the parkinsonian phenomena and produce a durable amelioration.

Davis, New York.

MUSCULAR INFANTILISM. Alexander Gibson, Arch. Int. Med. **27**:338 (March) 1921.

Gibson reports a case of unusual interest. In the family history of the patient there was definite hereditary evidence of similar conditions of muscular weakness. Of twenty-six males, fourteen were affected and of twenty-one females, seven were affected. The patient was unable to perform any unusual physical exertion from childhood to the date of his admission to the hospital at the age of 26. He got along very well in his work and could even play golf without discomfort, but anything more strenuous, like rowing or playing baseball, was impossible. On rising from a sitting position to an erect posture, he required the aid of his hands and arms. His general health and mental ability were always good. Physical examination revealed: height, 5 feet 8 inches; weight, 155 pounds; musculature small and poorly developed; distribution of fat rather in excess over the upper part of the trunk, over the abdomen and about the hips and thighs. His contour suggested the female type, with tapering extremities and small hands. The blood pressure was from 130 to 140 systolic; the blood picture was practically normal; roentgen-ray examination was negative. His bones were small. The Wassermann reaction was negative. There was no sexual impotence (nor was there any history of disturbance of reproductive power in the family).

He was given various organic extracts, some drugs and certain muscular exercises were prescribed. Raw suprarenal extract caused gastric instability and had to be discontinued. Graduated exercises caused no improvement. Thyroid extract made his general condition worse if it affected him at all. Pituitary gland feeding caused no improvement. Strychnin met with no response. His strength is well illustrated by a comparison made with a laboratory attendant who could do 400 kilogram cm. of work in two seconds with the second finger of the right hand, while the patient could accomplish only from 12 to 15—a percentage of the normal of 4 or 5. Microscopic examination showed a piece of muscle to be essentially normal.

Careful examinations of the urine were made in the laboratory, as well as chemical tests on blood. The conclusions arrived at were that the creatinin excreted suggested the actual extent of muscular power which he possessed. Creatin excretion suggested an infantile condition, and varied little on a meat diet. Blood sugar and sugar utilization were normal. On the basis of creatin in the urine (which is normally absent after maturity) and the small muscular power possessed, the author places the condition in the nomenclature of muscular infantilism.

Patten, Philadelphia.

THE FATE OF THE NEURAL CREST IN THE HEAD OF THE URODELES. F. L. Landacre, J. Comp. Neurol. **33**:1-43 (April 15) 1921.

Since Miss Platt's demonstration, in 1894, of the contribution of ectoderm to the formation of cartilages and other mesodermal structures in amphibian embryos, the origin and significance of this mesectoderm (i. e., mesoderm of ectodermal origin) have been subjects of much study and some controversy.

Landacre, in salamander embryos, has been able to follow the development of the ectodermal cells which form the neural crest of the head up to late stages when the tissues have acquired their definitive characteristics. In urodeles the neural crest is first incorporated into the neural tube, then is detached and rapidly extended ventrally. From the dorsal portion the general cutaneous and general visceral components of the fifth, seventh, ninth and tenth cranial ganglions are formed, together with some loose mesenchyme. The ventral portion of the continuous neural crest sheet of cells extends downward ultimately to the ventral surface of the head and gives rise to loose mesenchyme and to a number of the cartilages of the head, namely, the anterior portion of the trabeculae, Meckel's cartilage, the palatoquadrate bar and all of the branchial cartilages except the second basibranchial. The greater part of the mesenchyme in the ventral head region and in the branchial region is of ectodermal origin. The remaining mesodermal structures of the head (muscles, connective tissues, cartilages, etc.) are of entodermal origin.

The dogma of the specificity of the germ layers is thus apparently broken down. The significance of this two-fold origin of the mesoderm is not clear. Speculations regarding its possible relations to the origin of sympathetic ganglion cells, chromaffin tissue, neurilemma sheath cells, etc., must await on further observations.

C. J. Herrick, Chicago.

SINISTRALITY IN RELATION TO HIGH BLOOD PRESSURE AND DEFECTS OF SPEECH. Clarence Quinan, Arch. Int. Med. **27**:255 (Feb.) 1921.

The subject of sinistrality is taken up from the standpoint of heredity, prevalence and symptomatology. The author agrees with Rameley whom he quotes as follows: "The condition is a Mendelian recessive and probably exists in about one-sixth of the population." From recent statistics, it seems that about 4 per cent. of normal people are left-handed. In regard to the "master-eye" and sinistrality, one writer is quoted as finding 57 per cent. right-eyed persons who were left-handed. Speech defects are common in sinistrals as compared with dextrals, probably because there is more difficulty in finely coordinated movements in the former class. Also, sinistrality is exceptionally prevalent among the mentally defective. The author suggests as explanation that there is a relative inferiority in the organization of the central nervous system.

With regard to the blood pressure in sinistrals, a survey of 600 men was made living under the same conditions, the majority being over 60 years of age. Of this number, forty-two were sinistrals, and twenty-eight of these were left-handed, while fourteen were right-handed sinistrals (that is, those who by training have become right-handed but are by nature left-handed). Fifty per cent. of the forty-two were right-eyed and 50 per cent. left-eyed (classified according to the "fixing eye"). The fourteen right-handed sinistrals were left-eyed. For further classification the forty-two were divided into three groups: (1) left-handed, left-eyed (pure sinistrals); (2) left-handed, right-eyed (crossed sinistrals); (3) right-handed, left-eyed (crossed sinistrals). For comparison with sinistrals, blood pressure records were made on a group of 100 unselected right-handed, right-eyed dextrals, and for tabulation purposes this group is called Group 4 by the author. Five men in Group 4 as well as five men in Group 1, and two men in Group 2, gave a history of stammering. The average blood pressure values in the four groups were: (1) 166.7, (2) 182.1, (3) 174.9, and (4) 154.5 mm. This shows a percentage of from 71 to 85 of high pressures in the first three groups as against 46 per cent. of high pressure in Group 4. The percentage of stammerers in the four groups was: (1) 35.7, (2) 14.2, (3) 0, and (4) 5.

The author concludes that "high arterial tension occurs more frequently in left-handed than in right-handed people, and that it is suggestive of constitutional inferiority. Stammering occurs in sinistrals with a frequency of from three to seven times greater."

Patten, Philadelphia.

A MYOCLONIC TYPE OF MYELO-ENCEPHALITIS OF MALARIAL ETIOLOGY. M. G. Marinesco, Ann. de méd. **9**:1 (Jan.) 1921.

The writer first gives a clinical description of a case, in which fever, widespread myoclonus, prostration and death on the twenty-fifth day were the outstanding features. There were 10 cells in the spinal fluid. The case was considered as epidemic encephalitis until a subsequent blood smear showed the presence of malarial parasites.

The necropsy report, which is given in detail, establishes the etiologic significance of the malaria. In brief, the chief pathologic findings were the presence in the central nervous system of small so-called malarial nodules. Measuring from 210 to 132 microns, their histologic constitution depends on a leukocytic element combined with a cellular element of vascular origin. Cells carrying pigment are present and in some instances cells bearing the parasite. The presence of the latter and of the pigment justifies the term malarial nodule.

Marinesco considers the essential cause of their genesis to be a formative irritation of vascular origin.

Their distribution included the cerebral cortex, the peduncles and bulb and both the gray and white substances of the cervical and lumbar cords. The cerebellum had been invaded by them only to a slight degree and the spinal ganglions to still a less extent.

The writer points out especially that the nodules were not associated with hemorrhages. The latter were encountered in areas, free from nodules.

Though the involvement of the central nervous system was widespread, necropsy study failed to demonstrate malarial parasites in either the spleen, the liver, the suprarenals or ovaries.

Davis, New York.

ADMINISTRATION OF A PITUITARY EXTRACT AND HISTAMIN IN A CASE OF DIABETES INSIPIDUS. R. B. Gibson and F. T. Martin, Arch. Int. Med. **27**:351 (March) 1921.

The case is that of a man, 24 years of age, who complained of loss of appetite, pain in the back and abdomen, occasional dimness of vision, coldness and chills, polydipsia, polyuria, general weakness and loss of weight. The onset of these symptoms was indefinite and the polyuria could not be traced farther back than 1914, when he contracted syphilis. The bearing that syphilis has on the condition could not be definitely determined. The physical examination revealed no evidence of pituitary disease, except that the sella turcica was enlarged; this was not interpreted as abnormal. The blood pressure was: systolic, 104; diastolic, 62. The spinal fluid Wassermann reaction was negative, but the blood showed a + + + + reaction.

The laboratory work carried out in the case consisted of thorough studies of metabolism, with frequent examinations of the urine. At the time of admission the volume of urine varied from 15 to 20 liters per twenty-four hours and had a persistent low specific gravity. Hypodermic injection of pituitary extract (obstetric pituitrin) gave a response in a short time, but in reality the effects were fleeting. The ingestion of the tablet preparations gave a similar response, but its effects were more enduring. The total volume of urine was lowered to 10 liters or less per twenty-four hours, and the nitrogen output was considerably decreased. The percentage of uric acid was high, however (conforming to the reports of Hammett, Patten and Suitsu on nonendocrine cases). The nitrogen partition in general was normal; glycogenesis was not reduced, but there was a hypoglycemia.

Histamin injections produced results somewhat similar to the pituitary injections, but were much less effective. The systemic reactions to the preparation were so severe (intense headache and general prostration) that it was discontinued promptly. The authors conclude that histamin is not the active principle of the pituitary gland.

The general conclusions were that pituitary substance exerted only a temporary effect on the polyuria, but during its activity appreciably diminished the volume of urine, decreased the nitrogen output and increased the uric acid percentage.

Patten, Philadelphia.

Society Transactions

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Regular Monthly Meeting, April 21, 1921

James B. Ayer, M.D., *in the Chair*

IS THE TREATMENT OF PATIENTS WITH GENERAL PARALYSIS WORTH WHILE? Dr. Harry C. Solomon.

Opinions differ as to whether or no it is worth while to treat patients with general paralysis. To distinguish even pathologically between cerebrospinal syphilis and general paralysis is difficult. Cases that apparently are cerebrospinal syphilis prove to be general paralysis or vice versa. For a diagnosis of general paralysis, in addition to typical clinical findings there should be certain laboratory findings: a Wassermann reaction which is positive with 0.2 c.c. of spinal fluid, a paretic gold curve, globulin, excess albumin and lymphocytosis about 100. Practically all of the nine patients shown met these requirements; their treatment had consisted largely of the intensive use of arsphenamin, 0.6 gm. twice a week over a period of three or four months.

In the first case treatment was begun in 1916. The patient left the hospital after two months of treatment, apparently having clinically recovered. Her spinal fluid was practically negative when examined in 1919. It is now positive, but she has been clinically well for a period of five years.

The second patient was a man of 53, who had been admitted to the hospital with a diagnosis of general paresis in 1915. During the first four months he received a great many treatments with spinal fluid drainage. When last examined, January, 1921, he had a negative Wassermann reaction, a cell count of 7 and the colloidal gold curve was 0001100000. He is now making twice the salary he made when admitted.

The third patient did not do well under intravenous injections. He was admitted in April, 1916, aged 37. After two months' treatment with arsphenamin and mercury he was no better, and he was then given three intraventricular injections followed by seven intravenous injections of arsphenamin. On November 16 the spinal fluid was negative. The fourth patient was sent to the hospital because he had become inefficient. On March 14, 1919, he had a shock and felt dizzy when he got out of bed. His memory had been poor since then and he had speech defect and writing disorder. He has received both intravenous and intraspinal injections, and although his spinal fluid is not quite negative, he is back at work and is as efficient as he was in his best days.

The six other patients showed similar results. Dr. Solomon thought that if these patients had not been treated, they would have died or would still be at institutions instead of living for a period of from two to six years in good health and with good efficiency.

DISCUSSION

Dr. Arthur H. Ruggles said that cistern puncture had been used about fifty times at Butler Hospital with no bad results and with the most satisfactory cooperation on the part of the patients. They seem to have less reaction following this treatment than after the intraspinal method, and it presents a new point of attack in the case of patients who do not respond either to intravenous treatment plus spinal drainage or the combined intravenous and intraspinal treatment.

Dr. Solomon said he could not give the exact figures for the number of remissions in the patients treated. Some were treated only a short time because it was impossible to get cooperation. In 1916, of all the patients treated in the state hospitals 25 per cent. were allowed to go out on remissions. Age does not seem to play an important part. Patients treated at 65 years of age have done very well. It is impossible to say why certain persons do well and others badly. A young man with a good heart and kidneys may do poorly, possibly because of an overwhelming infection that has used up his immunity.

Regarding Dr. Ayer's method of cistern puncture, he thought it a practical addition to the treatment of these cases. It is revolutionary in the treatment of brain and cord inflammatory conditions and will greatly alter prognosis.

Dr. Solomon also advocated intravenous injections, if intensively continued, and considered one of his chief mistakes to have been overconservatism in giving no more than 0.6, 0.8 and 1 gm. to a patient. The worst that could happen as a result of too large a dose would be to shorten the life of a patient with only a few months to live, and it has never been shown that larger doses damage or approach the tolerance of the patient.

ENCEPHALITIS WITH SYMPTOMS INDICATING DISTURBANCE IN THE REGION OF THE PITUITARY GLAND. Dr. Douglas A. Thom.

Dr. Thom presented a young girl, born in 1902, who had never been sick until February, 1920. The family history was negative. She graduated from high school at the head of her class two months before the onset of her illness and took a position as a stenographer. Up to the time of her illness she was normal in every way, was not hypersensitive and had no character twists. On Feb. 4, 1920. she dreamed of an automobile accident in which her brother was hurt and she was killed. All the following day her mind was filled with the content of this dream. She went home anxious and worried to find that her brother was all right. From that time on she was restless and was unable to sleep for about a week. Then she slept continuously for four or five weeks. At the beginning of this drowsiness she had diplopia and strabismus. The drowsiness has persisted. She has been emotionally unstable since that time. At the present time she is well-developed, highly cooperative, talks rapidly without much expression, appears to be very intelligent and has perfect insight into her condition. She is strong and well but has a masklike expression; she is otherwise normal. The physical examination is negative except that the hands are held in the parkinsonian attitude, the shoulders droop slightly forward, and she is conscious of a tendency to fall forward. The Wassermann reaction is negative. She suffers from extreme thirst. While at home she drank ten quarts of water daily and excreted about the same amount of urine. This condition has persisted for about a year, dating from a few weeks after the onset of the lethargic condition.

The problem is whether the pituitary or neighboring region has been involved which would account for these symptoms of diabetes insipidus.

ENCEPHALITIS WITH PROMINENCE OF HALLUCINATORY SYMPTOMS. Dr. L. J. Thompson.

Dr. Thompson reported two cases of encephalitis with hallucinations unaccompanied by delirium. Hallucinations in encephalitis have been noted before, but in almost every case have been credited to the delirium. In these two cases, however, there has been no delirium. Of the eighteen patients with definite encephalitis with hallucinations treated at the Psychopathic Hospital, all had delirium except two, and one other.

The first patient was a woman, 37 years old, married, whose symptoms began in November, 1919, with dizzy spells; once she fell. Shortly after she began to have severe, transient, occipital headaches. In January, 1920, she began to have involuntary movements of the legs, arms and head, which shook continuously, so that she could hold nothing, and could neither dress nor feed herself. One month before entering the hospital, a year ago, her psychotic symptoms began. She became suspicious, and said that the people upstairs were taking moving pictures of her which were to be in the newspapers; that they were boring holes in the ceilings and pouring skunk oil through them; and that men were coming through the window. Several times she thought she saw her deceased husband; she said that there were soldiers around the bed. At one time a sword came up beside her bed and disappeared. At the hospital she continued to show hallucinations in all spheres.

Examination showed ptosis of both eyelids, irregular pupils and dancing movements of the eyeballs. There was no definite disturbance of reflexes, but general muscular hypertonus. The spinal fluid showed increase of globulin, 27 cells, a gold curve of 2334321000 and a negative Wassermann reaction. The hallucinations continued for a week or ten days and then suddenly disappeared; she had good insight. At that time the Parkinson syndrome began to increase. She had slight tremor of the hands, a masklike face and dysphagia increased. She left the hospital but still shows residual symptoms.

In the next case there may be some doubt as to the diagnosis. Last July slight shaking of the head was noticed. About three weeks later she said that certain people began to talk about her. Later she had definite hallucinations and was sent to the hospital. She had had headaches for a long time. The spinal fluid showed increase in globulin, 7 cells, and a negative Wassermann reaction. A recent lumbar puncture was negative. After being in the hospital a little while she developed ptosis of the left lid. The knee jerks were unequal. The patient has continued to have definite auditory hallucinations, to which she occasionally reacts by outbreaks of temper.

Dr. Percival Bailey said that during the past year, with Dr. Fritz Bremer, he had made a series of experiments on dogs which showed that the polyuria which often accompanies operations on, and diseases of, the pituitary gland is due to a lesion of the hypothalamus. The patient presented by Dr. Thom had a definite postencephalitic syndrome, but with polyuria excluded there were no pituitary symptoms. That polyuria is due to a brain lesion has been insisted on by Ochsner, Camus and Roussy, Houssay, and Leschke. Recently Harve has described the pituitary glands from several cases of epidemic encephalitis, and one familiar with the structure of that organ would recognize in his description nothing which could not be found in any pituitary gland. Recently, also, Maranon has described cases in which polyuria, adiposity and genital dystrophy have followed epidemic encephalitis.

Dr. Benjamin T. Burley said that in one instance only had he seen several people living in the same house attacked. He mentioned a case closely resembling the one reported by Dr. Thompson. The husband of this patient had died six months before in Worcester. He had been tapped, and the fluid was bloody. The patient had a fluid of the same content. A 16-year old son was taken sick four days after the mother, and he lived only seven days.

Dr. L. J. Thompson mentioned a necropsy case of encephalitis in which the patient had had visual hallucinations. Little was shown pathologically. There have been no cases at the hospital which might indicate contagiousness.

PRESENTATION OF A CASE OF PSYCHOSIS. Dr. C. Macfie Campbell.

Dr. Campbell demonstrated a patient with a psychosis, simple from the point of view of a mechanism, poorly represented in the textbooks and difficult to classify.

The patient was a girl of 24, who was well on March 3. On that day, at her work in a factory, another girl told her a great many things "a single girl should not know." She began to worry and felt queer, emotional and dizzy. She went out, was in a very excited state, wanted to take off her clothes and felt that she had to demonstrate her purity. She was brought to the hospital where she showed some knowledge of her whereabouts. All that night she stood up and looked out of the window; said God was cremating her and that she did not want to die in black. During the next four days she was very much disturbed; by the end of the week the excitement had passed away, her mind was clear and her convalescence had been uneventful.

The mechanism of the case seemed simple. The girl lived a quiet, reserved life, was very critical of approaches by any young man, lived rather more strictly than those around her, and was evidently very sensitive to the sexual topic. The conversation of another factory worker inflamed her imagination, and in the psychosis she lived out a drama of being kidnaped as a white slave. She saw the world in a distorted way and had odd fragmentary delusions, some of which were difficult to interpret. A year ago this girl had been rather upset by an Italian who was making advances to her. She came home very much excited and was emotional for two or three days. Then she passed into a condition in which she spoke little, and for two weeks she was looked on as a sick girl. She did not go to work for six weeks.

The case was a recurrent psychosis, not a simple hysterical delirium of the wish fulfilment type; there were a few elements of rather odd symbolism. Schizophrenic features were present, but the condition was benign and did not pass into a condition of deterioration.

The advisability of trying to make the patient realize the significance of the attack was discussed. This is a type of disorder which plays a large part in practical psychiatry, but which is, as a rule, discussed in a very inadequate way.

DISCUSSION

Dr. A. W. Stearns said that lately he had been much interested to note the ingenuity with which earlier psychiatrists used the assigned cause in diagnosing mental diseases. It is a question whether modern writers are going to prove as ingenious as their predecessors in making use of such material.

Dr. C. M. Campbell stated that he had not attempted to bring out the whole etiology. All that was known was that the girl was earning her living and working well until she left that morning for the factory. Naturally, the efficient cause of such a psychosis is a complicated group of forces. The patient's general efficiency was about that of her social group.

Tentative Classification of Behavior Disorders Used at the Psychopathic Laboratory, Massachusetts Reformatory, Concord, Mass.

	Responsibility	Manifestations	Groups	Diagnoses
Behavior disorders	Annulled	Alieniations	Psychoses Epilepsies	....
		Deficiencies	Intelligence defects	Idiot Imbecile Moron Subnormal
	Limited	Aberrations	Psychoneuroses Constitutional psychopathies Neurological disorders Endocrinopathies	....
	Entire	Habits of action	Characterial deviations or rectitude	

Diagnoses	Supernormal	Normal or Adult	Subnormal	Institutional
Psychoses				
Recoverable	......	......	......	......
Recurrent	......	......	......	......
Chronic without deterioration....	......	......	......	......
Chronic with deterioration.......	......	......	......	......
Traumatic	......	......	......	......
Alcoholic	......	......	......	......
Syphilitic	......	......	......	......
Senile	......	......	......	......
Arteriosclerotic	......	......	......	......
Brain tumors	......	......	......	......
Myxedematous	......	......	......	......
Epilepsies				
Grand mal	......	......	......	......
Petit mal	......	......	......	......
Jacksonian	......	......	......	......
Masked, etc.	......	......	......	......
Intelligence defects				
Idiot	......	......	......	......
Imbecile	......	......	......	......
Moron	......	......	......	......
Subnormal	......	......	......	......
Psychoneuroses				
Hysteria	......	......	......	......
Psychasthenia	......	......	......	......
Neurasthenia	......	......	......	......
Paranoid personality	......	......	......	......
Constitutional psychopathies				
Coordination psychopath	......	......	......	......
Sex psychopath	......	......	......	......
Neurological disorders				
Neurosyphilitic	......	......	......	......
Tremors, tics, chorea...........	......	......	......	......
Endocrinopathies				
Characterial deviations *				

* Specified in case notes and there associated, contrasted and illustrated. Certain observed deviations of frequent occurrence are the too egocentric, lacks self-respect, sex conflict, anti-social, lacks definite ambition, acquisitiveness unchecked, too liabile emotionally, unamenable to reason or authority, etc.

A METHOD OF PERSONALITY DIAGNOSIS AND EVALUATION WITH PROVISION FOR SOCIAL SERVICE PROPAGANDA. Dr. Guy C. Fernald.

Recent advances in the study of defective delinquents owe much to the findings in the field of character, that component of mentality which connotes the quality thereof, in contrast to its degree, i. e., intelligence. Those mental organizations eventuating in action or behavior are quite as significant as indexes of personality efficiency as are those mental organizations which eventuate in thought and its expression. In actual daily usage and in judicial procedure each personality is held accountable for his behavior, the product of character chiefly, and not his thinking, the product of intelligence.

Characterial deviations and rectitude may not be, as yet, technically tested and numerically scored but are susceptible of presentation by scientific description as are the findings of psychiatrists to the insane. The three fields of inquiry are mental disease, intelligence and character.

The outline is susceptible of such modifications as are required for adaptation to a given group and is submitted for its possibilities rather than as a compendium.

No form of presentation is complete which does not recognize the limitations of categorization. Search must be made in the character of a person for the strength of will needed for overcoming tendencies to sloth, which often defeat the success of those of high as well as those of a lower degree of intelligence. Failure, sociologically and economically is at least as often due to mediocrity of ability to continue to pursue a well selected purpose as to paucity of academic knowledge of the course to pursue. Of two imbeciles incapable of self-determination the one living happily and usefully on a farm as chore boy, trusted within limits but always supervised, the other of no higher grade of intelligence, a tramp who will beg or pilfer but will not work; the one does as well as he may, while the other does not. The essential difference lies in the field of character.

The psychiatric personality investigator's method of choice, then, is first to determine the presence or absence of mental disease, then to canvass the mentality for intelligence age level and for the determination of character deviations and rectitude, the last to be presented in the form of scientific description.

DISCUSSION

Dr. William Healy said that he was glad that Dr. Fernald was emphasizing the study of character as a thing by itself. Even in the study of persons from the standpoint of vocational possibilities, mental tests, which are nowadays so much advocated, are by no means sufficient. As a well-known silk manufacturer said, mental tests, except for clerical workers, did little in the way of giving him useful information in his large factories. And recently a paper has been published, based on investigations in this factory, in which it comes out clearly that there is little correlation between the behavior prognosis, that is, the success at work, and the results on mental tests in the factory at large. Some of their most reliable workmen are people of poor mental capacity. Indeed, it would seem that one would have to be pretty dull to be thoroughly happy pasting labels on boxes for eighteen years. Along this line was a case that Dr. Healy saw years ago. A young man appeared in court accompanied by his employer, who was anxious to pay the fine for a slight delinquency. He explained that the offender was the only one he had ever had who did his particular bit of work satisfactorily. The young fellow turned out to be definitely defective. The employment was in a laboratory, keeping things clean. Thus, there are necessary phases of industrial life in which intelligence is even a handicap to good performance. Character and temperament have a great deal to do with both good conduct and bad conduct. The point of view of Aristotle cannot be assented to; namely, that good intelligence would prevent a man from doing wrong.

But another trouble is that in making studies by means of so-called intelligence tests, all the elements of intelligence are not considered. This is coming out clearly in a symposium on intelligence tests that is appearing in the *Journal of Educational Psychology.* It seems to have taken psychologists a considerable time to grasp this point.

Dr. Fernald's categories are decidedly useful for a practical view of the situation, but Dr. Healy feels that the idea of responsibility is altogether too difficult to be placed among the categories. It is not open to exact thinking. What is responsibility? How can it be measured and recognized? It is a metaphysical conception which cannot be clearly defined, and it has nothing to do with the practical handling of many cases. It is time that psychologists and psychiatrists taught the practical issues of this question plainly to the legal profession. The law attempts to demand the answering of questions that cannot be answered.

Dr. Fernald, in closing, remarked on the importance of character in the life activities of an individual. Defective intelligence is stationary. It must be taken as it is and cannot be improved on, but character growth can be stimulated and improved as may be observed in social service endeavor.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Regular Monthly Meeting, May 19, 1921

James B. Ayer, M.D., *in the Chair*

An unmarried man, 30 years of age, whose family history was negative, had been well up to the time of the present trouble, with the exception of having gonorrhea in 1917. A Wassermann test made some time subsequently was negative. After his enlistment in 1917 he took long hikes in intensely hot weather. They were very exhausting and caused pain and numbness in the anterior portion of his right leg and in the right little toe. The pain and numbness would disappear on resting, but the leg slowly became weaker. After ten months, he had some sort of infection after which the leg was very weak. There had appeared a superficial scar about the size of a silver dollar, reddish blue in color. He was discharged in January, 1919. By this time the scar had spread somewhat and was numb, but not painful. He went to work on a buffing machine which created intense heat, and he noticed that when it touched the scar no heat was felt. The sensation of cold was probably also lost. At present the scar had spread considerably, the anterior thigh and leg muscles were weak and the right anterior thigh numb. There was pain in the anterior tibial group. There had been a suggestion of the same phenomena in the left leg.

The patient was well nourished and unusually muscular. The gait was not remarkable. The Romberg sign was absent. The pupils, fundi and facial muscles were normal. Lateral movements of the eyes caused two or three nystagmoid oscillations. The tongue, soft palate, spine, and muscles of the upper extremities, back and torso were normal. The patient said that the strength of the right upper extremity had diminished. The heart was normal. There was no atrophy of the lower extremities. Occasional fibrillation was observed in a very small area in the anterior tibial region. The general strength of the extremity was not commensurate with the muscle volume. All forms of sensibility were preserved except over the lower anterior aspect of the right thigh. Here, in a region roughly 3½ by 4 inches, was a keloid formation somewhat red and traversed in part by veins. Here, and for a short distance beyond its borders, thermal and pain sense were entirely abolished while tactile sense was partly lost. Muscle and joint sense, arm, tendon and peri-osteal reflexes were good. Knee and ankle jerks were present and equal but not very active. The abdominal reflexes were good. The scrotal reflex on the right was markedly exaggerated, plantar reflexes were present and equal but not lively.

The two outstanding features of the case were the low level of the cord lesion and its limitation to one posterior horn.

Dr. Courtney suggested the following pathogenesis: The hikes probably acted as a trauma, producing hematomyelia. Then followed gliosis, then a breakdown of the gliotic tissue and cavity formation. Oppenheim states that he has seen only three such cases. With regard to the prognosis Dr. Courtney mentioned a woman seen a number of years ago, in whom the disease was well developed. He saw her again ten years later. Meantime she had borne three children. The disease had made no progress.

THE PATHOLOGY OF TRIGEMINAL NEURALGIA. Dr. Percival Bailey.

The pathogenesis of trigeminal neuralgia is still unknown. In the days of peripheral operations examinations of avulsed nerves were made, but the only suggestive finding was the so-called "beading" of the myelin sheaths reported by Drs. Mitchell and Spiller (*J. Nerv. & Ment. Dis.* **25**:400, 1898). Later on, when the ganglion was removed, various reports of examinations appeared. Of these, only four were concerned with nerves not previously subjected to operative interference. Head (Allbutt's System of Medicine, vol. 8, p. 724), Monari (*Brun's Beiträge* **17**:495, 1895), and Coenen (*Arch. f. klin. Chir.* **67**: 333, 1902) reported the ganglions they examined to be entirely normal. Schwab (*Ann. Surg.* **38**:696, 1920) thought there was some increase in interstitial tissue. Probably this was an error in interpretation, but even if correct, it is an unessential finding because no changes were found in the nervous tissues.

During the past year I have examined eleven ganglions from Dr. Harvey Cushing's operations, in only one of which, however, had there been no previous operative interference. For comparison sections were available from fourteen other cases in which there had been no previous operative measures.

In the one previously unoperated case, E. P., Surg. No. 14016, aged 50, the ganglion was immediately placed in 96 per cent. alcohol and stained with thionin. The specimen might have served as a model for one of von Lenhassik's plates. The ganglion cells were perfect. Nor could any pathologic changes be seen in any of the other structures. Sections from fourteen other ganglions previously unoperated showed no marked pathologic change. Three of them were fixed in liquor formaldehydi and stained by Marchi's method; three were fixed in liquor formaldehydi and stained with hematoxylin eosin, and eight were fixed in Zenker's solution and stained with hematoxylin and eosin or methylene blue-eosin. In the Marchi specimens considerable fat was revealed but not resembling that of secondary degeneration. For the most part, it consisted of the fat which normally increases in the connective tissues of the aged and the dirty deposit to be found in any nervous tissue which has stood in liquor formaldehydi. There was no "beading" of the myelin sheaths as described by

Spiller. The hematoxylin and eosin specimens were examined especially for any possible increase in interstitial tissue, and none was found. In the interstitial tissue of spinal ganglions, as well as in the trigeminal, were found numerous collections of closely grouped nuclei which resembled round-cell infiltrations. They had no pathologic significance. In some of these patients at operation the arachnoid may be seen to be thickened and adherent, but the same may be said of aged persons without trigeminal neuralgia. We may conclude, then, that the ganglions from patients with trigeminal neuralgia are essentially normal.

Eleven ganglions were examined from patients who had previously been subjected to peripheral procedures. In them also the pathologic changes found were exceedingly meager. One was studied by Marchi's method; two by the Marchi-Mallory combination of Jacob and the Marchi-Mann combination of Alzheimer; four by Alzheimer's method V on frozen sections; two by Nissl's method, and two by Bielschowsky's method in frozen sections and Hassin's Bielschowsky-Mann combination.

In neither of the Nissl preparations were there any signs of reaction at a distance. The ganglion cells not crushed by the scissors were well formed, filled with tigroid, the nuclei centrally situated, and there was no increase in fat or pigment content. The Marchi preparations showed rarely the "balls" of material which stain with osmic acid, as Spiller has described, but counter-staining, according to Jacob or Alzheimer, showed absolutely no reactive phenomena on the part of the Schwann cells. This finding cannot be accounted for. The material, whatever it is, lies within the myelin sheaths. There are absolutely none of the phenomena of secondary degeneration. Alzheimer-Mann and Bielschowsky preparations rarely showed a swollen or distorted axon, and never a fragmented one.

We may conclude that, aside from senile changes in the connective tissue and parenchyma alike and occasionally reaction at a distance in the cells from peripheral operations, the ganglions from cases of trigeminal neuralgia are normal. The essential lesion in this disease must lie in the periphery, probably in the nerve endings (Harris: *Brit. M. J.*, May 22, 1920). That section of the nerves at the peripheral foramina stops the pain until the nerve again regenerates is sufficient to establish this point, and after peripheral operations there is no reason to expect any changes in the ganglion other than the well-known minor ones in the cells, for the phenomena of secondary degeneration occur peripheral to the section and not central. So far as I know, the nerve endings have never been examined. Initiation of the pain by cold air, which causes reflex constriction of the vessels of the skin, and by talking, during which the skin is irritated by superficial muscles, etc., indicates a peripheral origin.

DISCUSSION

Dr. Harvey Cushing said that the character of the pain, its unilateral situation and the manner of its spread, speak against the end organs as the seat of the lesion. Division of the nerves as well as alcoholic injections lead to certain changes in the ganglion which may be sufficient to inhibit for a time the discharge of paroxysms. It must be said, nevertheless, that trigeminal neuralgia is much more often bilateral than is commonly observed. About a year ago, all his gasserian patients were communicated with and replies were received from about 90 per cent. of the 360 patients. Possibly 4 or 5 per cent. of these patients after several years had begun to have pain on the other side. It is a curious thing that true neuralgia of this type proceeds by a definite march. It goes from first to second to third or from second to third to first; it never jumps from first to third or the reverse, which would imply that the process lies in the center rather than in the peripheral nerve endings.

Dr. J. W. Courtney saw confirmation of Dr. Bailey's conclusion in certain clinical facts. In a considerable number of cases of migraine, certain attacks were characterized by pain limited strictly to the first and second divisions of a fifth nerve, with temporary residual tenderness on pressure of these divisions. From this he argued that as migraine results from a vasomotor ataxia, it was not unlikely that the pain in tic douloureux was the result of vasoconstriction of the nutrient arteries of the trigeminal nerve. This notion appeared to him all the more tenable because genuine tic douloureux most commonly occurs in arteriosclerotic patients.

Dr. Cushing replied that patients with major neuralgia were often young people, some actually in their teens, and he instanced a typical example in a lad of 16 years. He said that many of these patients had some disorder of the teeth or infection of the sinuses. Some of them, too, had a history suggestive of migraine, but trigeminal neuralgia rarely starts in the brow. Probably 60 per cent. of the cases start in the second division, 30 per cent. in the third division and only about 10 per cent. in the first division. Where there is primary supraorbital pain, one must be very suspicious of its nature.

Dr. Bailey said that the usual march of symptoms from one branch to another was more consistent with a peripheral origin, for if the disease were central, one might expect more often involvement of all three branches. The fact that one gets only a small part of the ganglion is offset by the fact that the part always removed contains the cells of the third division which is most frequently involved.

Dr. Ayer's remark might be answered by noting that section of the nerve peripheral to the foramina will not stop the pain in syphilitic basilar meningitis. The origin of the pain is not identical with that of trigeminal neuralgia.

LUMINAL POISONING. Dr. A. H. Ruggles.

In 1913, the dosage of luminal was given as from 1½ grains up to 12 grains. In *The Journal of the American Medical Association,* May 17, 1913, Dr. F. J. Farnell reported two cases showing toxic symptoms after the administration of luminal as a hypnotic. As far as is known there has appeared in American literature no similar report of the toxic effects of this drug; hence the report of this case.

E. G., a woman, aged 39, married, had always been somewhat nervous, tended to worry and could not stand fatigue or excitement. Married five years, she had no children, on medical advice. Following influenza a year and a half ago, she became more nervous and she had been in bed most of the time since. Her chief complaint was sleeplessness, and for the nine months previous to her admission to Butler Hospital she had taken 3 grains of luminal every night, with the exception of eight days when she tried to get on without it. During that time she is said to have slept brokenly or not at all. She took 1½ grains of luminal at bedtime and when she woke up during the night. On admission to the hospital speech was slow and slurring and the face expressionless. There was incoordination of arms and legs, difficulty in standing and a slow, unsteady gait. Sensation was normal. There was a tremor of the lips. The pupils were large but normal. Deep reflexes were normal. Luminal was at once discontinued, but the disturbance of speech and gait continued for about two weeks. At the end of three weeks all the symptoms had disappeared, and the patient has recovered her former health.

This case seems to show that there is the possibility of toxic effects from the continued administration of luminal even in relatively small doses.

DISCUSSION

Dr. William F. Boos stated that luminal must be used with the greatest discretion, but if so used, it is very valuable. Its action is very much the same as that of other drugs of the barbituric acid group (barbital and derivatives). It is closely related to barbital, and the symptoms of poisoning are similar to those of barbital in poisoning, except that they are accentuated. The dose of luminal should never exceed 3 or at most 5 grains a day. A number of epileptic patients under treatment with luminal had never shown any toxic effects. At times there had been a slow, scanning speech, but that occurred when bromids were taken in addition to the luminal. The rash which is sometimes seen is brought on by the other preparations also. Dr. Boos had an interesting experience with barbital with a woman who was in the habit of getting very much intoxicated and who had lately been using perfume. She was given large doses of barbital, luminal, a little chloral and bromids. The next morning she awakened quite refreshed but complained of the doses being too homeopathic. During that day she drank more alcohol in various forms and became very much intoxicated. The next morning she sent to the drug store for a tube of ten barbital tablets. When seen not long after she had taken 35 grains in less than seven hours, and she showed symptoms like those of luminal poisoning. She appeared intoxicated, was unable to walk, spoke with difficulty, and her eyes had a peculiar dilated effect. Later she obtained three more barbital tablets, taking ten tablets in less than ten hours. The next morning apparently she was all right. Probably the after-effects of the alcohol acted as an antidote to the drug. Had she been a normal person, she might have been seriously ill, but as pain is an antidote to morphin, so the extreme nerve irritation that she suffered, the sleeplessness and restlessness and the sense of fear, were so potent that they succeeded in counteracting such a powerful drug as barbital. The effect on walking is marked in all these drugs.

The main point is that the action of luminal is very similar to that of all the derivatives of barbituric acid, except that it seems to be the most powerful.

A drug which can be strongly recommended is bromural. It is very much milder in its action and is practically harmless. It is also a derivative of urea. A young girl who attempted suicide took at one sitting more than fifty tablets. She went to sleep and slept for forty-eight hours, and then awakened refreshed. The correct dose is usually two 5-grain tablets every three hours.

Dr. Donald Gregg mentioned an epileptic patient who, under luminal treatment, became quite euphoric and pugnacious and much more troublesome than before. The epileptic attacks seemed to be abolished, but he became very hard to manage. There was no particular drowsiness or difficulty in walking.

Dr. Ruggles referred to a case of poisoning with this group of drugs in which the woman first took 90 grains of barbital sodium, then 110 grains of barbital and then 200 grains of barbital without any prolonged ill effects. There must be a good deal of individual idiosyncrasy in the reaction to these drugs, for there has been reported in the literature a case of death from 15 grains of barbital.

ANHEDONIA. Dr. A. Myerson.

A full abstract of this paper appeared in the July, 1921, issue of the Archives, page 101.

DISCUSSION

Dr. H. I. Gosline remarked that Dr. Myerson had taken certain symptoms from a number of diseases and had traced them back to certain psychologic categories. In introspective psychology the organic sensations form one of the fundamental groups. The next step would be to work back to the enteroceptive nerve fibers, the glands of internal secretion and all other anatomic entities capable of reaction and inhibition. Here is the beginning for a rational psychanalysis instead of an interpretative psychanalysis.

Dr. Myerson pointed out that the condition he had been describing was a syndrome. It may be traced back, as Dr. Gosline suggested, to a physiopsychologic and endocrine basis. All the psychoses may be traced to a physiologic basis. This condition occurs in many diseases, and it is the cardinal symptom in the manic depressive group. Whenever the energy content of the individual becomes disturbed, this condition tends to occur. It is a basic reaction of man to anything that interferes with his outward energy.

IS THE FACT OF A PSYCHOTIC PERSON BEING THE ELDEST OR YOUNGEST IN A FAMILY OF ETIOLOGIC IMPORTANCE? Dr. E. M. Pease.

A study was made of cases from the manic depressive and dementia praecox groups—forty from each, and an equal number from each sex in the two groups. These two psychoses were selected because they furnished 40.48 per cent. of all first admissions to the state hospitals of twelve different states during the year 1919. They also represent 25.80 per 100,000 of the general population.

As heredity forms one of the most important of the assigned predisposing causes of these two psychoses, a possible reason for this was assumed to be due in part, at least, either to immaturity of the gonadal plasm at the time of conception of the first child, or to the beginning of involutional regressive changes in the parent at the time of conception of the youngest child. A further suggestion is the possibility that the imbalance in the eldest child may be due to instability of the endocrine glands other than the gonads in parents of immature years or, in the case of the youngest child to atrophy or reduced function of the parental endocrine glands due to advancing years.

The group of cases studied was very small owing to incomplete data in many of the records consulted. To be more conclusive: The study should include the other psychoses, also a comparison with the corresponding group of families in which there have been no psychotic members. It is hoped that such data may be presented later.

The results of the study were: Of the whole series, 27.5 per cent. were the eldest and 18.75 per cent. the youngest children. The whole series also showed 30 per cent. of the males to be the eldest and 12.5 per cent. the youngest; of the females, 25 per cent. were the eldest and 25 per cent. the youngest. Grouped according to psychoses: In the manic depressive series, 32.5 per cent. were the eldest, and 15 per cent. the youngest children. Of these, 30 per cent. of the males were the eldest and 10 per cent. were the youngest, and of the females 35 per cent. were the eldest and 20 per cent. the youngest. In the dementia praecox series, 22.2 per cent. were the eldest and 22.2 per cent. were the youngest. Distributed according to sex: 30 per cent. of males were the eldest and 15 per cent. the youngest, and 15 per cent. of females were the eldest and 30 per cent. the youngest.

CHICAGO NEUROLOGICAL SOCIETY

Regular Meeting, March 17, 1921

G. B. Hassin, M.D., *President, Presiding.*

BEHAVIOR DIFFICULTIES. Dr. David M. Levy.

Dr. Levy reported the case of a patient, aged 16, with dementia-praecox on whom mental tests were made over a period of two years.

Family History.—The mother had been very religious, the patient said that both parents had died as saints. She talked in a rambling, distractible way, bringing in useless details, but she managed the household well. The father's mother had had senile psychosis. The patient was the youngest of seven siblings; the other were employed chiefly as unskilled laborers. She had suffered from asphyxia at birth which required much effort in resuscitation. There had been no convulsions or fainting spells. She was a restless sleeper, but there was no sleep walking or enuresis.

Physical Examination.—This revealed an asymmetric skull and body, moist, cyanotic hands and feet, female distribution of pubic hair and moderate diffuse enlargement of the thyroid gland; pulse, 80; height, 65 inches; weight, 102 pounds; absent conjunctival reflex, sluggish corneae. Areas of hyperesthesia recorded in an examination made in 1919 were no longer present. Otherwise the examination was negative.

Mental Condition.—The patient received the Stanford revision of the Binet-Simon tests at the ages of 14, 15 and 16. In each case supplementary tests requiring briefer time intervals were also used (Trabue sentence completion) and were within a year of the Binet age. The mental ages scored were 13 years 4 months, 10 years 3 months and 8 years 6 months, respectively. This marked deterioration was in keeping with observations made by his school teacher, who spent much time with him because of his difficulty with simple problems in arithmetic which he had done with ease in the previous year. She observed especially that his difficulty with numbers was in marked contrast with his ability to read and his evident appreciation of difficult literary passages. In all these tests cooperation was secured, and the amount of deterioration determined (about five years) may be considered fairly accurate.

Of significance also is the fact that psychotic symptoms were not elicited until the third committment to the Detention Home at the age of 16. At that time the patient developed delusions of persecution, saying the doctors were all after him, they were trying to kill him; also that the world was going to end in three days; that he heard voices in which those words were announced. In the week previous to the expression of fairly definite delusions there was expression merely of vague fears and worries; "I'm worrying all the time, but it's about nothing in particular." The patient was committed on each occasion because of "incorrigibility" or stealing.

In the mental examination reaction time was slow, sometimes suspicious of blocking. He spoke with a slow, monotonous, depressed voice. He kept to himself in the ward. His dreams were filled with ghosts and spirits. When he read he visualized the characters until he could see them all "as big as life" in front of him. "Brave by day, but quite a coward at night," said the mother, "trembling violently after he is told a ghost story."

Frequent masturbation had occurred with a few instances of homosexualism. Faulty attention was shown in his increasing forgetfulness when sent to the grocery on errands. His thefts, which were the cause of his commitment, always occurred when he was alone and on one occasion after worrying a great deal he returned a bicycle which he had stolen. He ran away, always alone. He never had "pals;" concerning the other boys, he said: "They always pick on me, call me names, always four to one against me."

At the time of the first examination the father's description of his behavior difficulties was: The boy was stubborn, impudent, tried to aggravate by constant argument, ran away when in difficulty; spent his money for playthings; if told to do something he would wait a long time and mockingly ask "What did you say?" (early negativism?). School progress was poor; he complained that his teachers were always assigning him too large lessons; he constantly used big words, "but he does not know what they mean," said the mother.

DISCUSSION

Dr. John Favill reviewed two cases of behavior difficulty diagnosed as psychopathic personality, egocentric type.

The first patient was a boy of 10 who stole, lied, smoked and chewed tobacco, was bad tempered, defiant, emotionally unstable and stubborn. Both parents were nervous. The home conditions were good except for an interfering grandmother. Physically there was a suspicion of mild hyperthyroidism. The intelligence quotient was 117.6.

The second patient was a boy of 10 who stole, lied, and was emotionally unstable and extremely stubborn. He had refused to confess his last theft for one week at home and a two hour interview at the Institute failed to bring out the truth. In the evening, however, he confessed to his parents. Heredity showed moderate nervousness in both parents and one older brother who stole for a while when a boy but who outgrew this habit. Home conditions were excellent. The physical findings were unimportant. The intelligence quotient was 139.1.

After obtaining the confidence of these boys and attempting by personal influence and suggestion to adjust their chief difficulties, good results have followed to date, in the first case for five months and in the second for three months.

These cases, selected from a large number of this type, some of which have been under observation and successful adjustment for much longer periods, appear to show the importance of personality factors over those of environment. They illustrate the fact that, with our modern understanding of the mental factors in behavior, the treatment by suggestion and by careful utilization of the circumstances in each case is sufficiently successful to encourage a wider application of this method.

INTELLIGENCE RATING IN THE PENAL INSTITUTIONS OF ILLINOIS. Dr. Herman M. Adler.

The results of the survery of the penitentiary at Joliet, briefly summarized, are:

The groups of native-born whites, foreign-born whites and negroes in the penitentiary rate somewhat higher in general intelligence than similar groups in the draft army. In each of these groups at the penitentiary there is a smaller percentage of men of inferior intelligence and a larger percentage of men of superior intelligence than was found in the same group in the army. Native-born whites in prison rate markedly higher as a group than foreign-born whites. Northern-born negroes rate higher than Southern-born. All whites as a group rate higher than negroes. There is little relationship between intelligence and the age of the prisoners.

Although all grades of intelligence are to be found among the different groups of criminals, there is a tendency among the groups to differ in general intelligence. Those in the fraud group possess the highest level of intelligence; this group is followed by the three groups, burglary, robbery and larceny; there is a low rating for murder, while sex crimes rate lowest in the scale of general intelligence.

There is a larger percentage of men of inferior mental ability in the groups of prisoners who have served five or more years at the penitentiary than among those who have served less than five years. Prisoners who have records of previous imprisonment as a group have a smaller percentage of men of inferior intelligence and a larger percentage of men of superior intelligence than the group of prisoners against whom there is no such record. There seems, therefore, to be a general tendency for recidivists to rate somewhat higher in intelligence than first offenders.

Of the group of men of inferior intelligence by far the great majority are negroes and foreign-born. Less than two-tenths of one per cent. of the entire prison population may be classified as imbecile according to their intelligence ratings. Sixteen per cent. of the population rated below 11 years mental age.

There is a close agreement betwen the results of the survey of the penitentiary at Joliet and the reformatory at Pontiac. The population of these two institutions represents the majority of the adult offenders in the state of Illinois whose crimes constitute the more serious offenses. The intelligence surveys of these institutions do not indicate that there is a close relationship between mental deficiency and delinquency among this class of delinquents.

DISCUSSION

Dr. Clarence Neymann, at the psychopathic hospital, was struck by the fact that those people who were so prone to get into difficulties with their surroundings and made the greatest amount of trouble were usually people of average intelligence and were not, strictly speaking, patients who were mentally defective. Such patients were usually brought in by the social service departments with the report that the patient spent his money in getting drunk or for morphin and did not support his family—perhaps supported another woman. Dr. Neymann always doubted the strict correlation of feeblemindedness and insanity with behavior difficulties. Some people are troublesome to the community on account of mental deficiency, but most of them are not feebleminded. People with psychopathic personalities are unusually quick to adapt themselves to institutions and always arouse the sympathy of the physicians, especially of the interns. They always tell a good story, promise never to repeat their offense, but on going out they do the very things they have been warned against, and are brought back to the institution—only to make the same promises again.

The drug cases, the chronic alcoholics, the men who do not support their families, the men who commit sex crimes or peculiar sexual acts, the men at war with their environment—these patients are usually neither insane nor feebleminded, but have psychopathic personalities. This point should be recognized, and one should not attempt to solve all behavior problems along the lines of feeblemindedness and insanity.

Dr. Julius Grinker agreed with Dr. Neymann that there is a conspicuous absence of so-called dementia praecox in the penal institutions. He compared the statements of Dr. Adler with those made by others that almost every juvenile delinquent brought into court has a case of dementia praecox.

Dr. Grinker thought it was gratifying to hear some one who had made a profound study of the subject honestly admit that studies have just been begun and that definite conclusions cannot be drawn from so few figures. Such studies as Dr. Adler is making must ultimately lead to definite conclusions and meanwhile have a tendency to stimulate further research.

Dr. Herman M. Adler (closing the discussion) said the answer to Dr. Reitman's question depended somewhat on the personalities involved in the situation. In Illinois the staff officers of the institutions have no administrative authority. Such authority was refused because the success of the officers depends on their not having such authority. They have no power to order a patient in or out of hospital, they cannot order "punishment" or "no punishment," but act simply in an advisory capacity. In the penitentiaries they have an advantage over the physician in the state hospital in that in the state hospital there is a staff of physicians, and the head of the hospital is a physician who knows more than any one else on the staff. An assistant makes a recommendation, but the superintendent *is the physician* and can carry it out or not, as he wishes. In the penitentiaries they are all lay officials, and the warden has full charge and can do anything he wishes. If the psychiatrist tells him a certain prisoner is demented and should not be punished the warden can let him go unpunished or not, as he wishes, but if the psychiatrist makes correct diagnoses and does his work well, the warden will not take the chance of disregarding the advice, especially if the physician makes his diagnoses in writing and sticks to them. There will not be many trials before his word is carried out. Dr. Adler thought it was encouraging to know that the state penitentiary at Joliet, which was known as one of the worst in the country so far as structure and habits are concerned, has done better than any other of the state institutions. There the staff actually amounts to something and it passes on the problems suggested. This has been brought about by the great decency of the institution as a whole and the fact that the psychiatric work has been done carefully and properly and no recommendations have been offered that were not justified. The warden has turned over practically all the staff work to the psychiatrist and the old-time guards, that they had been warned against, come to the psychiatrist and put their troubles before him and seek his advice in the handling of many of the prisoners. If a man does not obey promptly and adapt himself to all the rules quickly they think there is something wrong with him and ask the psychiatrist to look him over—which sometimes proves embarrassing when the psychiatrist cannot find anything wrong. Instead of having to force their services on the staff they are really a little "over sold," and are trying to hold back and find out the answers to some of the problems. The same holds true of conditions at Pontiac and St. Charles and Geneva.

Dr. Adler thought the crux of the matter is that they did well in diagnosing and classifying people, but when it comes to making good on some of the promises about treatment and prevention they are a little short. In his opinion it is time to stop some of the propaganda and do some constructive work.

PHILADELPHIA NEUROLOGICAL SOCIETY

March 25, 1921

George Wilson, M.D., *President*

A CASE IN WHICH SEVERE GENERAL ATAXIA DEVELOPED DURING THE COURSE OF ACUTE ENCEPHALITIS. Dr. William B. Cadwalader.

This case is presented in order to call attention to the tendency to recovery in a type of severe generalized ataxia that may develop in acute encephalitis occurring in children. In this case the type of encephalitis could not be determined. Although severe ataxia may continue long after the acute symptoms have subsided, there is a tendency to improvement, in some instances with complete recovery.

History.—C. B., 3 years old, was admitted to the Presbyterian Hospital, Nov. 7, 1919. He had been healthy until two days before admission, when he was suddenly seized with abdominal pain, severe occipital headache and vomiting. The following day he had a series of general convulsions in which the lower limbs were held strongly flexed and the upper limbs were rigidly extended.

Physical Examination.—The pupils were equal and the eyeballs moved normally, but there was moderate lateral nystagmus. Moderate rigidity of the neck was noted. The cranial nerves, thoracic and abdominal organs and the extremities seemed to be normal, though there was a tendency to flaccidity. The tendon reflexes were absent. On admission the temperature was 102 F., the pulse was 132 and the respirations were 22. Blood culture and blood count were negative. The spinal fluid was clear and contained 7 mononuclear cells per cubic millimeter. One week later there were 19 mononuclear cells, and on two other occasions 7 and 10 cells.

Course.—A diagnosis of meningo-encephalitis was made. On November 14, one week after onset, there was moderate incoordination of the upper extremities with marked flaccidity. The fever continued, varying from 99 to 101 F. The incoordination gradually increased and involved the muscles of the neck, trunk and limbs.

The general symptoms did not subside until March 31, 1920. At that time the incoordination was so general and so severe that the patient was unable to feed himself, unable to stand, or even sit up in bed without support. With every voluntary movement the limb was thrown about in a wildly ataxic manner. When at rest there was no tremor. The limbs were extremely hypotonic, but not paralyzed. Hearing was impaired and there seemed to be some mental deterioration.

At present the patient is still moderately ataxic, but can walk although he frequently falls. His mother states that he can go up and down stairs. The tendon reflexes are absent, vision is good and there is no longer nystagmus. The incoordination or ataxia seems to be progressively diminishing.

TWO CASES OF FAMILY SPASTIC PARAPLEGIA. Dr. N. W. Winkelman.

The mother of the patients is in good health but has external strabismus. The father has had tinnitus for fourteen years with neither dizziness nor deafness. There are four boys and three girls in the family. One boy died at the age of 1 year from an unknown cause. The elder of the patients, an 18-year old girl, born in Russia, was admitted to the University Hospital. Nov. 23, 1920, because of tremor and difficulty in walking. Three years ago it was noted that the patient had tremor of the hands, and a year later she began to walk on her toes with spastic gait. The trouble has gradually increased so that now she has much difficulty in walking. She has received six injections of arsphenamin but has shown no improvement. She was born by a breech presentation and was asphyxiated. She had the ordinary diseases of childhood.

Examination revealed a mentality decidedly below par. The pupils were equal, regular and reacted normally. There was weakness of the right external rectus muscle, but no nystagmus. The reflexes of the upper extremities were increased but equal. Slight spasticity of the upper extremities was present. Rapid movements were awkwardly performed. The knee jerks were much exaggerated, the right being greater than the left. There was no ankle clonus. but the Babinski reflex was present on the right with a suggestion of one on the left. The legs were stiff. No impairment of sensation was found and no atrophy. The laboratory findings were: albumin and hyaline casts in the urine, blood count normal, Wassermann reaction and spinal fluid negative.

The other patient, the 2-year old brother, had a normal birth. At the age of 5 months the child fell out of a coach and since that time had been cross-eyed. While the child was still very young the mother noted a little difficulty in spreading the child's legs. He began walking at 20 months but has never walked normally. He is unable to talk. For the first time, a month ago, he had seven spells in one day but has had none since.

Examination revealed a child backward mentally, with internal strabismus. The upper extremities were normal. There was slight spasticity of the adductors and less of other muscles of the legs with a slight tendency to drag the legs. The reflexes were increased but no ankle clonus or Babinski sign was present.

A CASE OF DECEREBRATE RIGIDITY. Dr. J. W. McConnell.

R. C., a boy of 5½ months, was born after an easy labor. Apparently the child was normal at birth, except for distortion of the head which quickly disappeared. In less than a month after birth unusual stiffness of the whole body and a tendency to what was called "shivering" was noticed. When awake the child was constantly crying or moaning as though in pain, but nursing or a bottle of water would quiet him for a short time.

He was brought to the dispensary at the University of Pennsylvania, March 17, 1921, at which time the notable features were extreme opisthotonos and stiffness of the whole body.

Examination revealed a well nourished child. Eye movements were normal; there was no nystagmus; the pupils were normal. The head was of normal size, the anterior fontanel was unusually large; the posterior one more easily determined than it should be. There was no paralysis. The striking feature was hypertonicity of the entire body. Legs and arms were held very stiffly. The arms were flexed at the elbow, overextended at the wrist, with fingers flexed tightly over the thumbs. There was extreme opisthotonos; the head retracted between the shoulders; the legs were in complete extension with overextension of the feet so that in the opisthotonic position the supports of the body were the head at about the posterior fontanel and the soles of the toes. Despite this extreme hypertonicity the limbs were frequently in tonic-clonic movement, intermittent as to occurrence, and affecting the limbs as a whole so that they had the appearance of being shaken in the air with the shoulder joint and hip joint as the points of fixation. During all this time the child seemed to be quite conscious and was crying or moaning, the sounds being synchronous with expiration. The respirations were jerky and irregular with sighing once to every ten respiratory movements. The preferred position was on one side, usually the right. When placed on the back the opisthotonos interfered with the child's ease, and almost immediately the body would

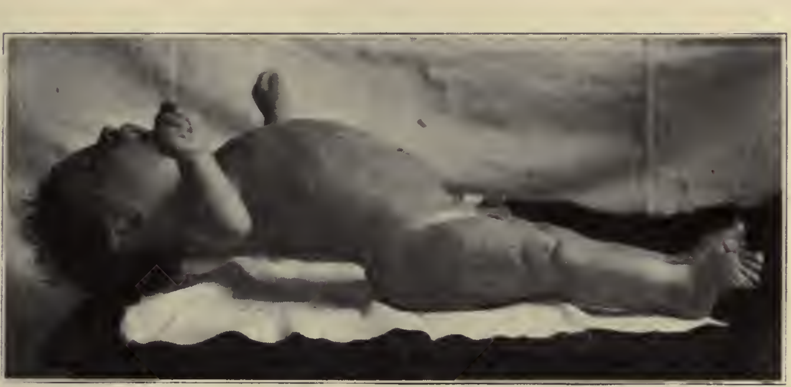

Fig. 1.—A case of decerebrate rigidity showing opisthotonos, retraction of the head, spasticity of the limbs, extreme pronation of the hands and extreme hyperextension of the feet.

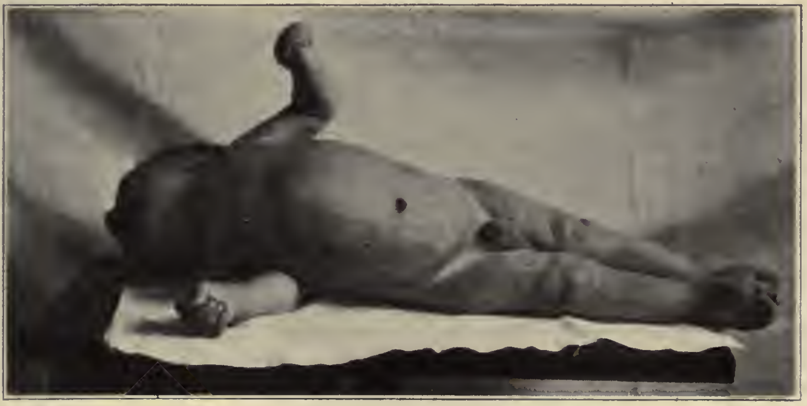

Fig. 2.—Case of decerebrate rigidity. The child is lying on his right side.

fall to the right side, rarely to the left. The hypertonicity was uninfluenced by position. Occasionally after gentle friction there was slight temporary relaxation. The intermittent tonic-clonic movements seemed to be uninfluenced by position or handling. A tentative diagnosis was made of decerebrate rigidity, with tonic so-called cerebellar type of convulsions.

DISCUSSION

Dr. W. G. Spiller said that the child presented a typical picture of decerebrate rigidity; the extension of the limbs and pronation of the hands were typical, but flexion of the toes was lacking. The condition was what Kinnier Wilson had described as decerebrate rigidity. Wilson called attention to the pronation of the hand in chorea and athetosis, and it was striking that many disorders presented more or less a picture of this condition. It was astonishing that this syndrome had waited so long for a description by Wilson or someone else, and it was noteworthy that Wilson had his first case in 1905 and did not report it until last year, and yet the clinical picture was so striking that no one could fail to recognize it.

Dr. J. Hendrie Lloyd asked why this case could not be an instance of posterior basilar meningitis, such as Barlow described and whether a spinal puncture had been made.

MICROSCOPIC SECTIONS FROM A CASE OF SYPHILIS OF THE EIGHTH NERVE. Dr. J. H. Lloyd.

This presentation was incorporated in a paper published in the May number of the Archives of Neurology and Psychiatry, pp. 572-579.

DISCUSSION

Dr. Charles K. Mills, referring to the possibility of the arsenical treatment causing deafness, reported the case of a boy about 15 years of age, who had acquired syphilis and who had, within a few months, developed symptoms of eighth nerve involvement and had become deaf rapidly. He, of course, was treated by the old fashioned method with full doses of mercury and iodid and yet went on to complete and irreparable deafness.

Dr. Francis X. Dercum believed Dr. Lloyd's case was one of syphilis of the eighth nerve. If deafness is caused by arsphenamin it is remarkable, considering the enormous use of the remedy, that deafness does not occur more frequently. Deafness in nervous syphilis occurs every now and then when arsphenamin has not been used. Such a case from Dr. Dercum's clinic was presented by Dr. Gilpin before the Neurological Society a few months ago. The man's condition subsequently decidedly improved as a result of arsphenamin treatment.

Dr. N. W. Winkelman said that they had not stained for spirochetes because the stain would stain axis cylinders, and if they were broken up, it was difficult to distinguish the spirochete from the axis cylinders.

Dr. H. S. Newcomer reported a patient, about 16 years of age, who received arsphenamin at irregular intervals from the time he developed secondary infection. After he had been under treatment for four or five months he developed deafness and dizziness, for which he was given weekly arsphenamin treatments and simultaneous intraspinal injections of mercurialized serum. At the end of about four weeks he had recovered from the deafness. There was a recurrence of dizziness but not of deafness which was similarly treated. For several years he has had no further symptoms.

Dr. J. Hendrie Lloyd said that others had observed cases like that of Dr. Newcomer, whose patient had developed deafness in the secondary period, and who had recovered from this deafness when the treatment was pushed. Dr. MacKenzie of Philadelphia, who had written the best paper on syphilitic deafness that Dr. Lloyd had ever read, said that they were not cases of arsenical poisoning; that a sufficient quantity of the drug had not been given. Dr. Lloyd said he had seen three cases of deafness develop rapidly after the administration of arsphenamin. The loss of hearing came on in a few days and was hopeless. He thought the cause was syphilis, but whether arsenic had something to do with it he did not know.

A CASE OF FRIEDREICH'S ATAXIA: EXHIBITION OF MICROSCOPIC SECTIONS. Drs. J. H. Lloyd and H. S. Newcomer.

This paper is published in full in this issue, p. 157.

DISCUSSION

Dr. N. W. Winkelman said that he had been very much interested in the sections. His first thought was that they resembled early tabes. The stained sections looked exactly like the drawings made by Trepinski years ago when he worked out the myelinization of the posterior columns. In the sections he saw the roots were normal. There was no doubt that in the lower cord the pyramidal tracts were affected and the cerebellar tracts higher up. Dr. Winkelman saw one section of cerebellar cortex which he thought was very much narrower. He did not think the granular layer had as many cells as a normal cerebellum. The Purkinje cells apparently were normal in number, but much decreased in size. They stained well. He would not have said that the cerebellum was normal.

Dr. W. G. Spiller said that the importance of this case was in the youth of the patient. The findings were valuable in showing the early lesions of Friedreich's ataxia. They brought up an important question: Are we to assume from the resemblance of the degeneration of the posterior columns to a certain stage of fetal medullation of these columns and to the degeneration of early tabes that this degeneration of the posterior columns in Friedreich's ataxia is exogenous in origin, i. e., that it begins outside of the cord? Dr. Spiller said that he had shown some years ago that many naked axis cylinders persist in the posterior columns, and by this fact he explained the preservation of sensation. The posterior columns in Dr. Lloyd's case showed typically the tourbillon described by Déjerine and Letulle, which had caused considerable discussion. He thought that one must be extremely careful in judging degeneration of Clarke's columns. The cells in these columns show changes more readily than almost any other cells of the spinal cord. Sections can be made through various parts of the thoracic region in normal cords without showing many of these cells. The region of greatest development of the columns, about the second lumbar segment, should be studied before one gives an opinion as to the scarcity of these cells.

Dr. J. Hendrie Lloyd said that the examinations showed that the cerebellum was normal, but he did not know whether the cerebellar peduncle had yet been examined. The large cells of Clarke's columns were destroyed at many levels. The inferior cerebellar peduncle consisted of six or seven sets of fibers; they were the connections with the cerebellum which one would think would be impaired. The cerebellum itself was not involved according to the opinions of all who had seen the sections. This cord was not syphilitic. The lesion was more than a posterior sclerosis. It was customary to speak of Friedreich's ataxia as a posterior sclerosis, although it is known that it is more than that. What the exact process was, Dr. Lloyd thought remained unsolved. It was a disease *sui generis,* of unknown causation.

Dr. H. S. Newcomer said he could not say what level of Clarke's column was examined. Six or more levels in the dorsal cord were examined. Most of them showed very few Clarke's cells and those which were present showed a great deal of sclerosis and simple chromatolysis. The tract sclerosis in the posterior columns was not of the tabetic type in which the fibers are laid down parallel to each other (columnar arrangement), the field being stippled with the ends of the fibers. The sclerosis here consisted of an irregular and interwoven mat of glia fibers.

SAN FRANCISCO NEUROLOGICAL SOCIETY

Regular Meeting, June 3, 1921

Milton B. Lennon, M.D., *President, in the Chair*

A CASE OF MYOTONIA ATROPHICA WITH ASSOCIATED BRADYCARDIA. Dr. M. H. Hirschfeld.

In addition to the classical findings in Thomsen's disease, this patient presented atrophy of the shoulder girdle muscles and periods of bradycardia.

History.—W. H., a young man, aged 20, whose family history was not known except that one sister had had a similar condition which had improved slightly, had attended school and had done general laboring. His habits and past history were negative. For as long a time as he could remember he had had his present difficulty in initiating movements. He felt muscle bound, but after performing a series of muscular movements he was able to proceed normally. Sudden fright or slight interference with his activities might throw his entire musculature into spasm and result in a fall. The malady involved the orbularis palpebrarae and muscles of mastication, but speech was not affected.

Examination.—He had a myopathic type of face. The shoulder girdle was atrophic, particularly the deltoids and rhomboids. Pectoral and biceps muscles were very small; they were unusually well developed in childhood. He had a wasp waist and lumbar lordosis, hypertrophied calves, quadriceps and glutei and active tendon reflexes.

All the body musculature, both hypertrophied and atrophic, showed the typical myotonic reflexes described by Erb, with the exception of the wave formation with stabile galvanic current, up to 20 milliamperes. However, slight variations in current strength produced contractions.

The basal metabolism, Wassermann reaction, lumbar puncture, blood sugar, and blood and urine creatin and creatinin showed no abnormality.

Course of Illness.—The day following the lumbar puncture bradycardia (40 beats per minute) developed and continued for one week. Under atropin the rate rose to 64. Electric cardiograms showed slowing of the entire cardiac cycle, particularly marked during ventricular systole. The patient gave a history of similar attacks with no known cause. Sections from the atrophic deltoid muscle and the hypertrophied glutei and vasti were normal.

DIFFERENTIAL PUPILLOSCOPE OF C. VON HESS. Dr. Otto Barkan.

With this instrument the intensity of light, which just suffices to cause the pupil to react, is accurately measured. This is read off on a scale and the figure used to express the "motor discriminative acuity" of the case in question. In normal cases a difference of from 95 to 100 suffices to cause pupil reaction. Comparison of the numerical values of the direct and consensual reactions, with other data, greatly assists in localization of the lesion in the reflex arc, even in the presence of optic atrophy or cataract. The method has proved of particular value in the diagnosis of incipient tabes, as by means of it a pupillary lesion can be definitely diagnosed long before any change in the pupil reaction can be detected with ordinary methods.

Book Reviews

PSYCHOLOGY AND PSYCHOTHERAPY. By WILLIAM BROWN, M.A., M.D. (OXON.), D.Sc. (LOND.), Reader in Psychology in the University of London (King's College), Clinical Assistant in Neurology, King's College Hospital, Late Medical Officer in Charge of Craiglockhart War Hospital for Neurasthenic Officers, Author of "The Essentials of Mental Measurement"; with a Foreword by WILLIAM ALDREN TURNER, C.B., M.D. Pp. 196. New York: Longmans, Green & Co.; London: Edward Arnold, 1921.

The bulldog is slow but tenacious. Probably England was the last civilized land to take a serious look at the psychoneuroses, and the war roughly taught her that she knew little of them. Having, with becoming deliberation, learned the lesson, she has now developed quite a crop of neuropsychiatrists or dynamic psychologists, and it would not be surprising a little later to find there the most uncompromising and dogmatic followers of Freud or Jung or Janet or Dubois, or a group of equally rigid psychopathologists of a distinctive English school.

The book of Dr. Brown, who is one of the younger English neurologists, is essentially a war product written by an honest, educated, thoughtful man who had a rich experience both just back of the front and in a neurologic service in England. Consequently it is a good book. But like many other authors who were decidedly immature when their war experience began and consequently had to take in a huge mass of information and experience in a too intensive fashion, Dr. Brown shows some evidence of indigestion and imperfect assimilation. Evidently he has tried to be broad-minded and catholic. Here and there he accepts more or less of the ideas of Charcot, Janet, Freud, Jung, Fierenczi, Dubois, Déjerine, Babinski, Prince, McDougal, Bergson and others. Indeed, as a composite picture—a bit blurred—of present day views of the psychoneuroses, the book will be interesting to all and decidedly instructive for the beginner, be he a young or old practitioner. Only two things are lacking; both can be corrected by time and experience. First, there is lack of self determination by the author. Except as regards dissociation and abreaction, he is not quite sure of himself. He grants too much to be entirely consistent. Second, the lesson of the war neuroses is not carried over into civil life.

The book is made up of five parts. The first part (two chapters, 35 pages) is introductory and briefly discusses the psychopathology of dissociation, multiple personality, psychocatharsis, the subconscious and the like. The second part (three chapters, 61 pages) is principally devoted to Freud's theories. The author distinctly disavows being a Freudian, but gives great weight to psychanalysis, 45 per cent. of the text being devoted to this subject. Part 3 is headed "Psychotherapy" (20 pages), but consists wholly of theoretical consideration of various factors operative in different methods. Part 4 (three chapters, 50 pages) is a discussion of the psychoneuroses of war, their nature, varieties and treatment. Chapter 9 is a useful chapter in which are compared cases of war neuroses seen early and those seen later. Just why the author should have tacked on Part 5 (a chapter of 22 pages) is not apparent. It is a rather discursive, somewhat immature and wholly useless disquisition on the relation of the mind to the brain, in which he finally is unwilling "to cast any doubt on the results of scientific investigation into spiritism."

The strong points of the work are the author's honesty with himself and the reader, his earnest attempt to get at the nature of the psychoneuroses and their best treatment, and his clarity of style.

The reviewer once heard an American jokingly assert to an English friend that in England crystallization is hereditary and congenital. As regards this little book of Dr. Brown, nothing could be farther from the truth. It is plastic almost to the point of confluence. Whether or no the reader accepts the author's conclusions, he has a fair and good presentation of the subject, a reasonable basis for opinions of his own.

The author is an implicit believer in abreaction or psychocatharsis in the treatment of hysteria and uses hypnotism to bring it about. Incidentally, it is interesting to note that many English neurologists have had to go through the evolutionary stage of hypnotism so long ago passed by other nationalities. Some of Dr. Brown's cases of abreaction in hypnosis are striking, and apparently his conviction of its scientific soundness is based on results of treatment—a rather shaky foundation when dealing with hysteria. Another interesting item is a collection of the histories of twenty-two patients treated in the field whose after-histories were obtained. All were treated with light hypnosis; only one had a relapse and fifteen returned to duty.

For long standing, intractable cases the author believes in what he calls autognosis. That is, by means of repeated explanatory, educative, persuasive talks the patient is given insight into his condition. Two cases are given in detail.

Perhaps partly because the book is to a considerable extent made up of discrete articles already published, it contains some careless statements and some contradictions. One or two of these may be mentioned. The unqualified statement is made that "hysterical patients are easily hypnotized." Of course, many of them are. Possibly every one of them could be hypnotized if all conditions, including the operator, were right, but certainly no one physician can hypnotize all hysterical patients.

On page 10 the author says that hysteria is "due to an emotional shock," whereas it probably never is. There are always preparatory mental processes, or there is mental elaboration following the shock. Indeed, the author himself later makes this plain.

Speaking of a (blindfolded) hysterical patient who said "yes" each time a normal area was touched and "no" each time an anesthetic area was touched, the author says "There is dissociated from the *entire mind* (italics ours) one psycho-physical power, in this particular case cutaneous sensitivity."

A pitifully small bibliography and a good index conclude the volume.

THE OXFORD MEDICINE. By Various Authors; Edited by HENRY A. CHRISTIAN, A.M., M.D., Hersey Professor of the Theory and Practice of Physic, Harvard University; Physician-in-Chief to the Peter Bent Brigham Hospital, Boston; and SIR JAMES MACKENZIE, M.D., F.R.C.P., LL.D., F. R. S., Consulting Physician to the London Hospital, and Director of the Clinical Institute, St. Andrew, Scotland. In Six Volumes, Illustrated. Volume 3, Diseases of the Digestive System, Kidneys and Ductless Glands. Pp. 828. New York: Oxford University Press.

This volume discusses diseases of the gastro-intestinal tract, the liver and pancreas, the kidneys and the ductless glands, except the thyroid; an article on this gland, by Plummer, is to appear later. It is a pleasure to be able to say that this volume maintains the standard set by the second volume. While a carping critic might point out some typographical errors and some curious English, such for example as a "reasonable reason," omitting the customary "if you know what I mean," these are not of sufficient moment to lessen the value of the articles.

As might be expected, the first article is on diseases of the esophagus, by Frothingham. The article is quite conventional and occupies a space proportionate to the importance of these diseases, but one might ask why, in a brief chapter, space is given to diseases of no clinical interest that might far better be given to the few important diseases of the esophagus.

Rehfus follows with a discussion of diseases of the stomach, excluding gastric ulcer. With proper force he points out the frequency with which gastric symptoms appear in a wide variety of diseases, a fact frequently forgotten not only by the general practitioner but also by the gastro-enterologist: "It behooves the physician therefore to treat every gastric case as one which is fraught with not merely possibilities of gastric disease and to recognize that even more frequently gastric disease is the expression of some systemic fault." Bad English, but an important clinical fact.

The discussion of the physiology of the stomach and of the methods of clinical examination is adequate.

It would be well for many general practitioners to read with especial care the chapter on gastro-enteroptosis, that they may avoid making this diagnosis as often as they do. Some of our surgically inclined friends could do the same with considerable profit.

Gastric carcinoma and gastric syphilis are well handled, but it seems to the reviewer that the author has given less discussion to functional disturbances than their frequency would warrant. We believe that gastro-enterologists are too much inclined to neglect cases of this type, or more properly speaking, to look on them as organic diseases. They overlook the patient in their interest in the examination of his gastro-intestinal tract.

In the chapter on ulcer of the stomach and the duodenum, Sippy again expresses his conviction as to the supreme value of alkalies in the treatment of these cases, and apparently his assurance increases as his already large experience grows. He leaves one in doubt as to his opinion on the importance of focal infections in the causation of ulcers and in their resistance to healing, but mentions in the paragraphs on treatment that foci of infections should be sought and removed.

His plan of treatment remains essentially unchanged and his indications for surgical treatment should be carefully read by all surgeons, especially by those whose writings suggest that any one who does not operate immediately after making a diagnosis of gastric or duodenal ulcer approaches the class of the criminal moron.

Stockton has written a carefully prepared, possibly too long, article on diseases of the intestines, with what appears to us to be marked disproportions in the distribution of space. This however is a matter in part of personal opinion, and he could no doubt justify giving nearly as much time to a discussion of the interesting but infrequent Hirschsprung's disease as to the common and troublesome chronic colitis.

Rolleston's articles on diseases of the liver and biliary tracts, illustrate the fact that generally the English are more careful in their writing than are Americans. While style, no doubt, is less important than material, a fact well stated makes more impression and is pleasanter to read than one badly stated. His discussion of the various types of jaundice is satisfactory, containing clear statements on a matter about which there is much confusion of thought.

Christian's chapters on nephritis are worthy of especial praise. They are clear and clean cut and gain much by his use of illustrative cases. The modern methods of testing renal function are adequately considered, and his classification of nephritis is satisfactory because it is clearly only a working basis, easily subject to such revision as must come with increasing knowledge. It is a relief to see a classification not based on pure speculation and not including fifty-seven varieties. This is a thoroughly good article.

The chapters on the suprarenals and especially on the pituitary gland are too sketchy and appear to have been abridged at the last moment.

ORTHOPEDIC SURGERY OF INJURIES. By VARIOUS AUTHORS. Edited by SIR ROBERT JONES, K.B.E., C.B., F.R.C.S., Director of Orthopedics, St. Thomas' Hospital, Surgeon, Royal National Orthopedic Hospital, Consulting Orthopedic Surgeon, Royal Infirmary, Liverpool, Honorary Adviser to the Ministry of Pensions (Orthopedic Surgery). Two volumes. Pp. 512 and 657. London: Henry Frowde, Hodder & Stoughton, 1921.

Volume 1 is devoted to deformities due to injury and gives one the distinct impression that it was intended to be a military manual. The material is almost entirely from military sources, and two full chapters are devoted to a discussion of team work between the casualty clearing station and the base hospital. Another is entitled "Splinting of War Fractures," and another is given over to the work done in centers for the limbless.

There is an excellent chapter on "Orthopedic Surgery of the Hand and Wrist" by Walter I. Baldwin. This contains much that is of great value in reducing the disability from this class of injury. A chapter by Sir Robert Jones on the relation of deformity to ankylosis and stiff joints is valuable. He gives clear instructions as to the positions of choice, as to breaking up adhesions, when to interfere and when not to, and gives his reasons for his beliefs.

The chapters on fractures, malunion and ununited fractures add nothing to recent developments along these lines. The book is an exposition of the methods of treating war injuries. It contains much valuable material which can be adjusted to the treatment of injuries in civil life.

The greater part of volume 2 is devoted to consideration of injuries of the central and peripheral nervous systems. Injuries of the head and of the spine are discussed by E. Farquhar Buzzard and Percy Sargent. The subject matter is good, but the space devoted to these subjects is so limited that one who is neither a neurologist nor a neurosurgeon will be little benefited by these chapters.

The anatomy of the peripheral nerves, to which the first chapter is devoted, is adequately described by A. Melville Paterson—obviously an abridgement of his monograph on the same subject. Many of the illustrations have been borrowed from standard textbooks on anatomy.

The chapter on diagnosis of injuries of the peripheral nerves by T. Granger Stewart and W. Rowley Bristow is well illustrated and fairly comprehensive. One useless feature is the inclusion word for word of the anatomic description of each of the nerves as it has already appeared in chapter 1 under anatomy of the peripheral nerves. This is mere padding and uses space which could be utilized for fuller clinical description.

An exceedingly well written chapter on the operative treatment of war injuries of the peripheral spinal nerves is presented by Sir Harold J. Stiles. It deals adequately with the surgical anatomy of the peripheral nerves and is very well illustrated.

There are two chapters on the end-results of nerve injuries and operations. These are valuable, but the space occupied by the tables does not appear to be justified by the lesson they teach.

A chapter on purely functional and reflex disabilities in their relation to orthopedic surgery is presented by L. leF. Burrow and William Cuthbert Morton. Some of the statements in this chapter probably will receive criticism at the hands of neurologists. For example, "orthopedic cases suffering from purely functional disabilities are seldom hysterical" and "there is reason to believe that any muscle which is ignored or suppressed may in time pass beyond the control of the will, possibly because it has become anesthetic through disuse." Again under treatment they state that "no patient is ever treated as a hysteric or malingerer."

"Trick movements" in cases of nerve lesions are very well described by Fredrick Wood Jones, and by means of double exposure photographs well illustrated.

The continuity of the material could, perhaps, be improved by placing the subjects dealing with diagnosis of peripheral nerve lesions, such as trick movements, next to the chapter on the clinical consideration of injuries to the peripheral nerves and placing the chapters on injuries of the head and spine after the chapters on peripheral nerves.

With few exceptions, the material included in this volume represents careful and accurate study of the types of injuries observed in the late war.

THOUGHTS OF A PSYCHIATRIST ON THE WAR AND AFTER.

William A. White, M.D. Pp. 137. New York: Paul B. Hoeber.

Dr. White, whose competence in the field of constructive psychiatry is known through his "Principles of Mental Hygiene" and "Mechanisms of Character Formations," has presented in this small compass the important comments of the psychiatrist both on the psychology that leads to war, and on the conditions which war superinduces and leaves behind. He presents the individual as a bundle of instincts that seek his own preservation and that of the species. He shows him existing, however, in a society necessary to his own life and training him to its own larger purposes. The process by which the organization of the individual into the social organism takes place is through repression of the individual instincts, so far as these run counter to the social functions, and their sublimation into socially valuable activities. Suppression takes place through the "herd critique," sublimation through "the utilization of the energies so repressed to find satisfaction in ways that are progressively more and more removed from primitive types." "The force that deflects the primitive instincts of love and hate from immediate satisfaction . . . is that group of necessities which arise in consequence of man's living together in groups, the so-called instinct of gregariousness, or herd instinct. "To put it another way: The force which, in its negative aspect makes for repression—the herd critique—produces in its positive aspect the desire for the reward of social esteem. As individuals are organized into groups, so are groups and communities organized into larger communities, nations and international groups. This takes place by a process of differentiation of function and organ. The process of this differentiation is away from primitive instincts and their immediate expression. Let the organization break down, and the primitive 'infantile' instinct reaches expression. Thus wars arise in the breakdown of international organization, and provide the opportunity for rebuilding, 'rejuvenesce,' an opportunity which has been necessary in the past. It remains to be seen whether a community of nations can be built up in the future which will be able to grow without this process of destruction precedent to rebuilding." The principle theme of the book is the display of primitive instincts, as the nature of the individual, and relatively unconscious, over against the social nature which has been subject to the training of the group. With any breakdown in this organization appear the primitive instincts, in what may be called their infantile form. This unconscious group of instincts the author considers selfish, and their appearance is in varying degrees, abnormal and degenerative. With full recognition of the opportunities which war presents for the self-sacrificing impulses and the higher ideals, Dr. White shows that in large degree war must, with its breakdown of the larger social organization, throw men back on the primitive instincts and set free that original human animal that is freed from the social restraints and group critique which makes of us civilized men. He lays stress on hate and fear as the attitudes which here predominate and which are antisocial in their influence. His comments on the expressions of these attitudes are illuminating and will have most salutary influence where read.

There is one conception largely used by Dr. White on which the reviewer cannot forbear a critical comment, and that is sublimation. In the necessarily summary statement in so short a treatise, sublimation appears simply as repression plus the turning of the energies of the repressed instincts into other and socially valuable channels. He thus leaves the reader with the impression, which is otherwise borne out, that man remains at bottom a primitive savage with a group of trained social habits. If these habits break down, the original savage appears. Is not this an inadequate psychologic analysis? Does it not present the training as an entirely external affair? What the reviewer feels this psychology overlooks is the fact that all real training is the result of a conflict of impulses, which leads to reconstruction and reorganization of the individual's own nature. It is only in so far as this inner reorganization is inadequately carried through that man remains at the core a savage. Human nature does not always remain the same. What our Freudian psychology still lacks is an adequate study of the growth of self. But this comment should in no way detract from the impression of the fundamentally sane and highly important message which Dr. White has given out of his professional experience and his experience of the war.

THE ENDOCRINES. By Samuel Wyllis Bandler, M.D., Professor of Gynecology in the New York Post-Graduate School and Hospital. Price, $7.00. Pp. 486. Philadelphia and London: W. B. Saunders Company, 1921.

In stating his opinion of the book, the reviewer is tempted to borrow some sentences recently used by the Cleveland physiologist, G. W. Stewart (*Endocrinology* **5**:299 [May] 1921), in discussing a certain article: "In reading this paper and many others by 'clinical endocrinologists,' the physiologist can scarcely escape the feeling that here he has broken through into an uncanny fourth dimension of medicine, where the familiar canons and methods of scientific criticism are become foolishness, where fact and hypothesis are habitually confounded, and 'nothing is but what is not.'" Equally apropos is

Stewart's comment on the contrast between the desert in which the physiologists and experimental pathologists have wandered and "the exuberant land of clinical endocrinology, flowing with blandest milk and honey almost suspiciously sweet." Granting that clinical medicine has a perfect right to run ahead of physiology if it can, such an advance is not promoted by almost delirious indulgence in wild hypotheses, of which this book is a product. Those who may be under the psychanalytic spell will here find another world philosophy with its phraseology and also a new basis for interpretation of dreams. We are told that dreams of being frightened mean overstimulation of the suprarenal medulla, dreams of fighting mean overactivity of the suprarenal cortex while dreams of making a speech are due to the anterior pituitary lobe. Childhood impressions and fairy tales are also of importance in Bandler's philosophy on account of their endocrine bearings. He shows that the Freudians are not the only ones who can elaborate a system of symbols. His endocrine symbolism bids fair to outdo theirs both in span and in obscurity.

SYPHILIS UND NERVENSYSTEM. Ein Handbuch in Zwanzig Vorlesungen für Praktische Arzte, Neurologen und Syphilologen. Von Dr. Max Nonne, Oberarzt am Allgemeinen Krankenhaus Hamburg-Eppendorf A. O. Professor für Neurologie an der Hamburgischen Universität und Leiter der Universitats-Nervenklinik. Vierte, Neu Durchgesehene und Vermehrte Auflage. Pp. 1019, with 169 illustrations. Berlin: S. Karger, 1921.

The first edition of this book having appeared twenty years ago, this, the fourth, edition requires little more than a book notice. For many years it has been a standard work and probably is familiar to every reader of the Archives. The author has made a consistent effort to bring the various phases of the subject quite up to date, and in this he has very well succeeded although, as he states in the preface, unable fully to consult recent literature outside of Germany. In places this lack is so noticeable as to be a real defect. Among the newer questions considered he mentions the curability of general paresis, syphilis of the third generation, family syphilis, the influence of the war on syphilis and the nervous system, and investigations relating to the spinal fluid.

Although this edition is more than twice as large as the first, the division into "lectures" has been retained but the number has been increased from seventeen to twenty. Obviously, the aim of the author has been methodically to cover the entire subject, and this he has done. No aspect of the subject is omitted. Indeed, occasionally there is pedantic fulness at the expense of definiteness. But here and there something is omitted. In a rather full discussion of prophylaxis, including consideration of castration of defectives and criminals, no mention is made of prophylactic applications following illicit coitus.

To his task the author has brought an unusual experience, great industry in assembling the findings and opinions of others and an open mind. An extensive bibliography, an index of authors and an excellent general index close the volume, which should have a place in every neurologic and psychiatric library.

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THE NEUROLOGIC DILEMMA*

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The opening chapter of William James' "Pragmatism" is called "The Present Dilemma in Philosophy." In place of philosophy read neurology, and the title of this president's address is revealed. The minor differences between James' chapter and mine will be found in the fact that I propose to devote just twenty minutes to the presentation of the dilemma in neurology. The major difference I shall refrain from mentioning, because I want to feel for a few moments, at least, worthy of the honor you have conferred on me.

It is far from my purpose to suggest a pragmatic solution, however, but rather do I want to certify to the existence of a dilemma and then to ask you to note that its partial solution can be found in the present program which my paper so feebly initiates.

Dilemmas exist only when attention is directed to them or when something happens which disturbs the orderly working of events and an effort is made to understand why things are not as they once were. I began to be dimly aware that there was something the matter with neurology when it was brought to my notice that in the neuropsychiatric service of the American Expeditionary Forces there were only a few neurologists, and that about 90 per cent. of the service was made up of asylum or institutional men. The war neuroses were essentially problems of neurology, and neurologists as a whole had very little to do with the attempt at solution. Whatever the explanation turned out to be is now of little importance, but this extraordinary lack of neurologic participation in one of the most significant neurologic opportunities of this generation led irresistibly to the conclusion that there were counteracting and antagonistic tendencies in neurology. It is out of these that the dilemma I am now going to talk about originates. It is my purpose then to touch briefly on the present state of neurology, to indicate what neurology seems to me to be, and to dissolve the dilemma by stating the things that make it up. As this address is not to be open to discussion, now at any rate, I shall find it comparatively easy to state such thoughts on the subject as I happen to have with a degree of boldness and definiteness not usually characteristic of presidents' addresses in our time and in our country.

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*President's address read at the annual meeting of the American Neurological Association, Atlantic City, June, 1921.

A BROADER CONCEPTION OF NEUROLCGY

Neurology just now presents a most curious and interesting anomaly, owing to the fact that the personal and clinical treasures of the neurologist are really no longer his. He is permitted to have only a far distant and often impersonal interest in them. One by one the old and classic standbys, the dead ends and fruitless scars of ancient processes are dealt with by those to whom a neurologic training and type of thought have been denied. Syphilis of the nervous system, tabetic and paretic reactions are all now well within the territory preempted by surgeons, dermatologists and internists. They make the diagnosis, plan and carry out treatment, often with skill and intelligence it must be admitted, without the least idea that they are treading upon ground discovered and cultivated by a preceding generation of intrepid neurologic thinkers. I mention this merely to indicate the steadily narrowing fields of activity left to those whose neurologic horizon is limited by the traditions of the past; traditions which are crumbling under the attacks of our more venturesome colleagues. I shall leave to your imagination the rest of this story. Rare spinal cord lesions, the curious and unusual types of disease, familial deviations, congenital anomalies upon whose unending diagnostic and therapeutic difficulties the neurologic intelligence wages a hopeless struggle, are the residuals left after the piratical sifting is finished. These may well be called dead ends in contrast to the live and vivid realities which a broad conception of neurology should encourage us to put our teeth into. I am leading perhaps somewhat deviously to the first point which I want to make, and that is that neurology must be redefined and we must be courageous enough to follow where this redefinition leads. Neurology is concerned with the totality of the problems presented by the nervous system in health or disease and any condition influenced by its disturbed function. Consciousness is perhaps the chiefest of these functions, and to its expression, whether normal or abnormal in the accepted meaning of that term, neurology and neurologists must be actively concerned. This points directly to that group of defects which neurologists have hitherto been only half-heartedly interested in—the psychoses. I am not referring to the legal and custodial phases, but to the direct participation in the study, care of, and direction of the insanities as they are met with in the daily experience of all of us. I desire to accentuate the conclusion that there is no psychiatric point of view, that there is no such thing as a psychiatric method of thinking or approach which logically can be divorced from the neurologic one.

It is the latter that contain the vital elements of scientific advancement. When the problems of consciousness are neurologically approached then progress will begin.

THE NEUROLOGIC POINT OF VIEW

What is meant, then, by the neurologic point of view? It is briefly this: What knowledge there is of disease of the peripheral nerve, spinal cord, and brain based on lesional defects was arrived at through anatomic and physiologic methods of thinking applied to conditions found in the nervous system. A mass of definite, tangible, and illuminating information was acquired, which, crystallized by observation, experience and thought, furnished the data of neurology. This attracted the attention and awakened the interest of neurologists from the period of S. Weir Mitchell and Charcot, roughly speaking, to the time of the war. The methods that grew out of their activity and out of the work of many other men of their stamp stood the critical test and were productive in establishing what is now in many respects a well correlated and reasonably well known field of knowledge. Such methods should be followed, modified in ways that can only now be surmised, when the problems are less tangible, less material, and, unfortunately, more complex.

The abnormalities of consciousness, expressed clinically in psychoses, become the objectives of neurologic endeavor as the logical development of the neurologic method reaches out to its ultimate possibilities. I am of course speaking of methods of thought, not technic of examination. The certainty of these conclusions appears to rest upon the fact that there is no fundamental difference in intellectual processes which can be said to be dependent on differences which are innate in the object of inquiry. The human mind in its function of thinking is not modified and does not undergo a change because the subject under scrutiny possesses one quality or another. The assumption that this is necessary causes the dilemma which has so long kept the neurologic method focused so largely upon purely anatomic problems. There is no fundamental difference in scientific method in the attempt to work out the factors that go to the making of a diagnosis of tabes, for example, and that which is concerned in manic depressive psychosis, dementia praecox, or a neurosis. If the method differs, and when it departs from the physiologic, anatomic, or chemical type of reasoning, then there is a defect in the thought process, and defective diagnostic and other conclusions are the result.

SENSORY DATA

In using the terms physiologic, anatomic, and chemical, I should like to include also such psychologic methods as are based on sensory data. This, however, by no means necessitates limiting in any way the sources out of which the essential data comes; but it is necessary, I think, to measure and weigh such data by the critical and objective qualities of neurologic thought, in opposition to the mystic or introspective methods which are deemed so essential when neurology is thought of as a domain limited and bounded by the peripheral nervous system on the one hand and tumors of the brain on the other. I fancy that the neurologist's objection to a great deal of the freudian conception of the neuroses is not due to the conclusion arrived at but to the loose methods of thinking indulged in. The assumption that a thing is correct because the expected is proved is much less convincing neurologically than if the unexpected sometimes happens. The sexual etiology of the neuroses does not strike terror to the neurogolic heart, but it is the prophecy and expectation that nothing else can be the explanation that is annoying and irritating.

There is here detected a faulty method of thinking which arises on account of the insistent and initiatively controlled trend which is wholly at variance with the free play of the trained intelligence upon a difficult and intricate problem. Psychanalysis as a method of investigation is possibly adequate, but it ceases to be so the minute it departs, in any important way, from the influence exerted by the deductive procedure developed in the building up of a neurologic method. In the investigation of the neuroses the same method of inquiry must be followed. These phenomena of an altered function of the nervous system are capable of investigation by the same methods of thought that are used when the object of inquiry happens to be a localized lesion of the spinal cord. The control exercised by the postmortem study is unfortunately lacking in the former of these, but in the clinical problem no difference in the method of approach is necessary. Causation, mechanism, and clinical end results are there just the same, even if the final test of the microscope is at present lacking. After all, the method is the main concern, and the scrutiny of final conclusions is to be governed accordingly. In the physiology of the nervous system is to be found the key to the neuroses, and from it will eventually arise the laws governing the grouping, distribution, and quality of the sensory objectivation of hysteria, just as definitely set as those produced by a tabetic lesion of the posterior root and column.

Please do not think I am advocating a physiologic parallelism. I have no definite ideas on this subject whatsoever. I am merely calling attention to methods of thought and ways of thinking.

It is obvious, therefore, that the field of neurology must of necessity embrace that aspect of deviation of the nervous system produced by anatomically demonstrated changes in its structure, that disturbance in its correct function that is called a neurosis, and that failure in adaptation which is termed a psychosis. This, I believe, is the field of neurology. This wide embrace is necessitated by the fact that there is no definite dividing line between them, and that the best and most fruitful method of approach is that which has had the best success in the understanding of the more obvious and more tangible problems, those that belong to the anatomic group.

THE NEUROLOGIC DILEMMA

I have tried to lay bare the elements out of which the dilemma in neurology has arisen. The dilemma, then, is this: The neurologist is caught in the meshes of contradictory tendencies in the subject by which its limits in one direction are steadily receding and those in another are extending beyond his visible horizon. He lacks the courage to wander into territory preempted by what appears to be a defective method because he lacks the conviction and faith in the correct method. He is too readily impressed by the conventional and traditional belief that some mysterious quality of mind is needed in order to venture into a land which by virtue of his training and experience he is best fitted to explore. I am convinced that the psychiatric phase of neurology, if such a paradoxical expression be permitted, is a definite part of any neurologist's activity and that the old separation of the subject should be given up. This does not by any means imply that there need be any limitations of specialists' activities in any direction. The neurologist may well choose one or another field or one or another group in which he is particularly interested. The whole, however, should be neurologically directed and infiltrated by the best neurologic thought. The solution of this dilemma must come about by keeping in mind very steadily this fact: neurology suffers when it separates itself too far from the problems of medicine as a whole. Psychiatry has, I believe, been artificially separated therefrom, and by its separation and segregation progress has been made unusually and uselessly difficult. The closer both get to the problems of medicine, the greater the advance will be, and if both do this they will automatically merge into the common subject to which they belong, which is disease of the nervous system. The place for neurology in the conception that I have in mind is a general hospital, where it should find its physical home. From this its material comes, and out of this combination its progress will develop. In a practical way, therefore, the university neurologic clinic will arise, as it is beginning to show itself in a few places scattered throughout this country. The totality of the problems of the nervous system will become, then, the object of inquiry, and the subject of this inquiry will be found in the persons whose nervous systems are diseased.

If it once becomes possible to view the nervous system as a whole, not only in its normal condition, but also when it becomes the subject of disturbing factors which produce what is called disease, the dilemma that confronts the neurologist will surely disappear, because it is the lack of a convincing method of approach that gives the dilemma its vitality, not the innate difficulties of the position itself. The impasse of a dilemma is usually brought about by the contradiction of tendencies, a pull in diverse directions. A stabilizing of effort or a neuteralization of energy results, and the thing that can be solved remains unsolved. In an attempt to rationalize this lack of progress the difficulties are assigned to a place where they do not belong, and a terminological defense system arises, which perversely enough becomes the dilemma itself. Against the false screen the efforts at solution are directed, and no wonder they so often fail. It is a fortunate thing that neurology has been able to build up for itself a scientific method that has all the characteristics of workability and adaptation.

Contrasted to this is a method of thought that derives from a prescientific era, in which what was inside of one's mind rather than the data outside was made use of. I make use of this rather crude comparison to point out the striking contrast between what I conceive to be the opposing thought tendencies in the two branches of nervous diseases to which the horns of our dilemma are directed. That this divergence of thinking exists can be easily proved by noting the mental behavior of a medical student in the presence of a case of tabes and a case of dementia praecox. The ease with which the former is examined and the awkwardness and embarrassment in the presence of the latter are striking indications of what I mean. As soon as a case of paresis is presented to him and it is indicated that the process here is identical with that of tabes, the contrast of thinking almost immediately disappears. The inclination to cling to the objectivation of symptoms and to feel a certain sense of comfort in the thing seen or felt is an almost pathetic reminder of the artificially produced breakdown of the neurologic method of thinking in the face of symptoms arising from disturbed mental functions. This is where I have tried to put my finger on the sore place out of which the dilemma arises.

THE TECHNIC OF MENTAL TESTS

It is the unwarranted assumption that a different kind of thinking is necessary in the one problem and its sudden realization that produces a blocking of smooth effort in the observation and noting of data and the necessary conclusion from them. The technic of a student in a case of dementia preacox in its neurologic, medical, and psychical phases is an interesting commentary on the clash of methods. The contrast to my mind should not exist at all. The use of material methods of observation as contrasted with those to which the term psychic is given is certainly striking. When the physical, neurologic, and chemical tests are over the examiner pauses as though to say, "Now I am going to examine this patient's brain or mind." Immediately there is a change in his attitude, a change which I believe is detrimental to such knowledge as he is able to obtain. What I am particularly interested in noting, however, is that with this change of attitude really goes no important fundamental change in method, because when it comes to the point of making a mental test or mental investigation or mental analysis, use is made and can only be made of the same channels of study that were used in the examining of the reflexes, or sensory tests, or what not.

After all is swept aside and all the hypertechnical phrases are done away with, there remains the method of question and answer. The instruments by which this is accomplished is by speech and by hearing. Everything else is adventitious, and what goes on in the patient's mind and comes into the examiner's mind does so through the sensory channel alone. It is the receptivity of one mind in the face of the attempted expression of the content in another's that satisfies all of the legitimate demands of the examination into the psychic field. Technic consists, if there is any such thing as technic in this sense, in the use of analysis, judgment, memory, the weighing of evidence, the use of past experience, the use of observation of facial expression, the reaction quality of emotion, etc., and if one examines this mass of data and the other mass of data he is not aware that there is any distinction of any importance whatsoever. The dilemma here consists in not admitting frankly and honestly that the difference in method is merely a difference in terminology; and, if it were not for the fact that terminological emphasis is placed on one method to the exclusion of the other, the neurologist's thought processes in one would be exactly the same as in the other. It is when the neurologist is influenced by the dilemma that arises in such an instance that the method itself begins to suffer, and unconsciously there is substituted something which is called psychiatric, and with which he feels unfamiliar because he instinctively knows that it is wrong. What I mean to imply is simply this: So far as psychiatric method is effective and productive it shares with neurologic methods a correct system of thought; when it departs it is manifestly defective. The dilemma here arises out of the impulse to dodge the hard issues of the neurologic pathway and to follow that part of the psychiatric method which falsely promises a way out. The curious thing is that it is this false element to which the term psychiatric is found most often applied.

CONCLUSION

The dilemma as it shows itself to me is made up, as most dilemmas are, of a series of false ideas, of illusionary elements, magnified into a semblance of reality by a terminology that finds a certain amount of attractiveness in its tautological nuances. Its chief incentive toward escape is based upon the false notion that because a thing is expected, all things are wrong that do not lead to that expectation.

The dilemma in neurology disappears as soon as it is faced or as soon as it is understood. The way out lies in the utilization of neurologic methods of thinking. The neurologist's opportunity is the nervous system, and no problem there is less than his right of possession nor more than his capacity, modestly and courageously tempered, may not hope to solve. Thus the dilemma vanishes and if, in attempting to dissolve it, I have not been able to state my thoughts with the grace and felicity of the great philosopher whose title I have dared to borrow, yet I hope I have been touched a little by his honesty of purpose and his supreme intellectual courage.

Humboldt Building.

INTRACRANIAL TELANGIECTASIS

REPORT OF TWO CASES *

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The two following cases of probable cerebral telangiectasis seem to be of sufficient interest to warrant report.

REPORT OF CASES

CASE 1.—*History*.—A merchant, aged 21 years, was referred by Dr. Ransom of Hancock, Minn., July 14, 1920. The patient's family history was negative except that his father died of diabetes. When 7 years old, he was thrown from a horse and struck on his head, but he remembered no details of the accident; appendectomy was performed on him when he was 14; he denied venereal disease. When 8 years old, while convalescing from an attack of mumps, he had a severe convulsion and remained unconscious for fourteen hours. He recovered and was well for three weeks, when he had another mild attack. Since that time the number of attacks have varied from three in one day to one in six months. He described a typical attack as follows: It begins with a sensation of electricity in the left hand, after which he feels as if the hand were off. Within a few minutes, he notices a jerking of the fingers of the left hand, gradually involving the muscles of the entire left arm. When it reaches the shoulder, he becomes semiconscious and feels as if he were floating in space. During this semiconscious period, he has excruciating pain in the left arm. Then he loses consciousness for a period of from three minutes to two hours. When he recovers consciousness he is confused and talks irrationally for a short time and his left arm feels weak and awkward for several hours. He has abortive attacks during which the hand becomes numb and the arm jerks, without loss of consciousness. Frequently he can arrest severe attacks by placing a rubber band tightly around the left arm. For the past four years he has had a constant feeling of numbness in the left hand. He does not have headache.

Examination.—The neurologic examination was negative except that he had impaired tactile sense of the left hand and decrease of abdominal and cremasteric reflexes on the left side. The fundi were normal. There was no telangiectasis on the face or body. Examination made three days later when he was complaining of indefinite general disagreeable feelings, revealed in addition a positive left Babinski and Gordon reflex and left ankle clonus. These findings were variable. The laboratory findings were: hemoglobin, 98 per cent.; leukocytes, 7,800; red blood cells, 4,480,000; blood pressure, — 110 systolic, 55 diastolic; urine, normal. The blood Wassermann reaction was negative. Spinal Fluid: pressure somewhat increased, no globulin, 4 lymphocytes, negative Wassermann reaction and colloidal gold curve.

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* Read before the Minnesota Academy of Medicine, April 14, 1921.

Diagnosis.—A diagnosis of jacksonian epilepsy due to irritation of the right motor region, cause undetermined, was made and craniotomy was performed Aug. 2, 1920, by Dr. Harry Ritchie.

Treatment and Course.—Enormously dilated blood vessels were found on the pia over the right motor region, arranged in the form of an irregular circle. These were ligated in four places and the dura was closed. During the next twenty-four hours the patient had twelve convulsions of such severity that chloroform had to be administered. The following night he had two more and was then placed on two-thirds grain of luminal three times a day. Up to the present time he has had only one unconscious seizure but at about weekly intervals he has had twitching of the left arm with blurred vision lasting a few minutes.

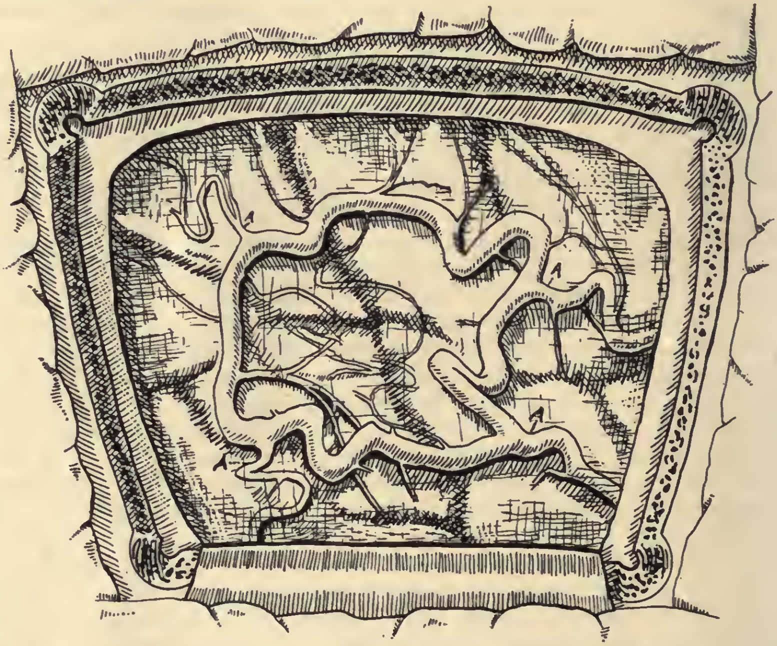

Fig. 1 (Case 1).—Jacksonian epilepsy; anterior upper margin of right ear. The vessels were ligated at *A, A, A, A;* × 1.3 +.

Comment.—Sachs reports two similar cases and believes that the clinical picture is sufficiently typical to enable one to arrive at a definite diagnosis. The outstanding symptoms are jacksonian epilepsy at long intervals in a nonsyphilitic, with unconsciousness of long duration, no evidence of intracranial pressure, slow progression of symptoms and telangiectasis on the head or face. With proper surgical interference, he makes an optimistic prognosis. Our patient conforms fairly well to this clinical picture, with one important exception—there was no evidence of superficial telangiectasis. In one of Sach's cases, the angiomatous process was found in the dura with numerous connections with the pial vessels.

Case 2.—*History.*—Our second case was that of a farmer, 26 years old, first seen in consultation with Dr. Robert Earl, July 8, 1915. His family history was negative except that the grandfather, mother and one sister suffered from periodic headaches. His personal history was unimportant except for an occasional headache prior to the accident. In July, 1914, while running, he struck the top of his head against a protruding heating pipe. He was unconscious for thirty minutes and was sent to a hospital where he remained for two weeks, during which time he was somewhat stuporous. Recovery was complete but ten months later he developed headache in the posterior midfrontal region. This headache was progressive, more severe in the evening and so distressing that the patient threatened suicide. Within one month he developed definite mental symptoms—he thought that people were talking about him and plotting against him and he threatened to harm them. He was also somewhat resistive and at times most unreasonable. Sleep and his general condition were satisfactory.

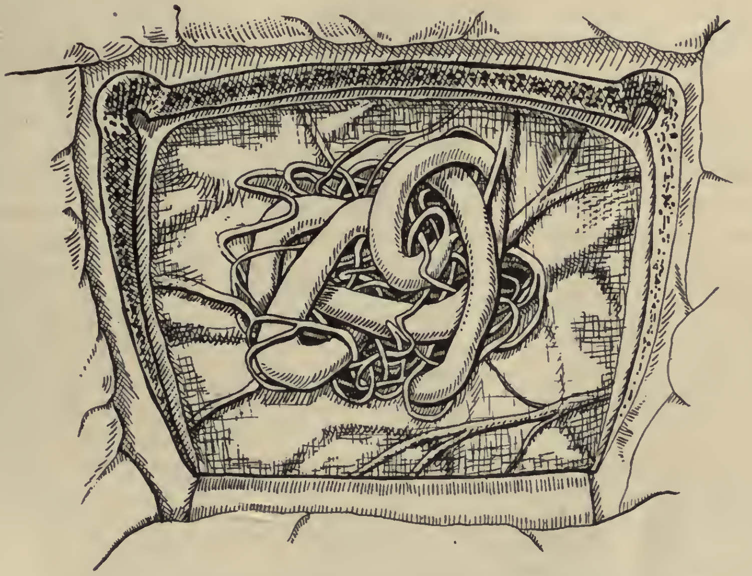

Fig. 2 (Case 2).—Multiple head injuries; inner canthus of the right eye; × 1.3 +.

Examination.—Neurologic examination made one year after the accident was negative throughout except that both knee jerks were sluggish but normal on reenforcement; there was a small area of marked tenderness to pressure over the posterior portion of the right frontal region. All laboratory findings were normal. The spinal fluid was under slight pressure, the globulin test was negative, 5 lymphocytes per cubic millimeter; the Wassermann test and colloidal gold curve were negative. A roentgen-ray examination of the head was negative.

Diagnosis.—A diagnosis of beginning dementia praecox or of a cyst secondary to the trauma in the right frontal region was made.

Treatment and Course.—Because of extreme tenderness over this area, operation was advised. Dr. Robert Earl made a right frontal bone flap and found the dura adherent to the skull. The dura appeared bluish, which was found to be due to a marked angiomatous condition on the surface of the brain cortex. This mass of blood vessels covered the entire operation field and appeared like a nest of bluish angleworms. Vessels were ligated in several places; bleeding was profuse but easily controlled. The patient made an uneventful recovery both physically and mentally.

Subsequent History.—The subsequent history is of interest. On Aug. 30, 1916, he was struck on the head by a small plank, which rendered him dizzy for a short time; after this he had occasional headaches. On June 6, 1917, he was blown up by an explosion, striking his head forcibly against a stone wall. His headaches were decidedly worse after that. On July 30, 1917, he stood for a time bareheaded in the hot sun, after which his headaches were so severe that large doses of morphin were required. Since October, 1916, spicules of bone had at times come through a discharging sinus at the site of operation. He was seen again Aug. 6, 1917. All neurologic findings were negative; the spinal fluid was normal. The old bone flap was loose at the median line and could be pressed in. On Aug. 8, 1917, Dr. Robert Earl transplanted two tibial grafts to fill the defect in the skull; the dura appeared normal except that there were extensive hemorrhages. He recovered and joined the Navy in November, 1917. In February, 1918, he was accidentally kicked on the head by another sailor and was unconscious for fifteen minutes, after which he recovered. It was found that he had a small depressed fracture over the right posterior frontal region. This depressed fracture was probably one of the tibial grafts which had become loosened by the kick. Ten days later, he developed generalized convulsions, as many as three in one week. During one of these attacks he threatened to kill the orderly with a razor. He was discharged from the Navy because of traumatic epilepsy. The skull wound discharged for about a year and then healed. During the year 1918 he had two more generalized convulsions. In July, 1919, he was again struck on the head by a small tree and was unconscious for several hours, after which he became acutely maniacal for two hours, during which time he attempted to kill one of the men with him. It required five men to put him in restraint. He recovered within an hour and remained in the hospital for two weeks when he returned to work. In July, 1920, he developed left foot drop and some frontal headaches. These continued until October when the foot drop improved, but the headache became more severe and memory was impaired. He was operated on Jan. 27, 1921, by Dr. J. A. Caldwell, United States Public Health Surgeon, at Cincinnati. The patient stated that the doctor had found depressed bone, removed it and filled the gap with a piece of rib. He has been perfectly well since that time, except for slight impairment of memory and the scars on his head. The neurologic examination on March 12, 1921, was negative except that the knee jerks were still sluggish, especially the left one.

LITERATURE ON THE SUBJECT

A true psychosis is not infrequent with cerebral lesions, especially when they involve the frontal region. Any form of mental picture may develop secondary to trauma. Under the head of post-traumatic constitution, Adolph Meyer[1] mentions paranoid states somewhat similar to the psychosis presented by our patient. The tendency to homicidal impulses in this case, on three separate occasions, secondary to trauma, is of rather infrequent occurrence.

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[1] Meyer, Adolph: Am. J. Insan., January, 1904.

Both Sachs [2] and Spiller,[3] in their articles on cerebral telangiectasis, give a fairly complete review of the literature. In the majority of instances, patients presented evidences of telangiectasis on the face, head or somewhere on the body. In the discussion of Sachs' paper cases were reported by Starr, Angell, Gordon and Hunt.[4] Steiner [5] reported the cases of three families with hereditary hemorrhagic telangiectasis and reviewed twenty-five cases from the literature. The outstanding features were telangiectasis and repeated epistaxis. In none of his cases, however, was any mention made of cerebral involvement. Our two patients had no superficial telangiectasis themselves and knew of none in relatives.

Cerebral angiomas have been classed as congenital tumors, new growths or secondary to trauma. When associated with telangiectasis elsewhere, the congenital factor seems an important one. In reviewing the cases in the literature, trauma is of frequent occurrence. According to Sachs, in certain cases it seems highly probable that a trauma has either irritated a preexisting growth making it develop rapidly, or has actually been the cause of the new growth. Local inflammatory reaction, such as adhesions, connective tissue overgrowth or roughened inner layer of the dura over the dilated vessels, are further evidences of this. The history in my second case and the operative findings lead one to believe that trauma probably was an etiologic factor.

Surgery is the only treatment for these patients. Ligation of the dilated blood vessels has given surprising results in some instances. Sachs suggests that the blood vessels be ligated in such a way that no thrombosis forms, but that they collapse after ligation, thus preventing irritation of the cortex. Spiller calls attention to the danger of extensive ligation and reports two cases. In one case hemorrhage occurred into the brain and in the other hemiplegia resulted.

In our first case, the four main trunks were ligated without any serious after-effects except the series of convulsions within twelve hours after operation, in which the patient almost collapsed.[6]

435 Hamm Building.

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[2] Sachs, E.: Am. J. Med. Sc., October, 1915.

[3] Spiller, W. G.: Congenital Tumor of the Brain (Telangiectasis) and Associated Cerebral Movements, Arch. Neurol: & Psychiat. **2**:50 (July) 1919.

[4] Nerv. & Ment. Dis., 1915, p. 634.

[5] Steiner, W. R.: Telangiectasia, Arch. Int. Med. **19**:194 (Feb.) 1917.

[6] In addition to the references given, the following may be of interest:
Cassiver, H.: Neurol. Centralbl., 1902, p. 32; 1910, p. 456.
Deetz, E.: Virchows Arch. f. path. Anat. **168**:341.
Kalischer, S.: Berl. klin. Wchnschr., 1897, p. 1059.

THE CONTRAST BETWEEN THE BRAIN LESIONS PRODUCED BY LEAD AND OTHER INORGANIC POISONS AND THOSE CAUSED BY EPIDEMIC ENCEPHALITIS*

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Brain lesions caused by inorganic poisons, such as lead, are collectively designated encephalitis. The latter term is also used for cerebral lesions caused by infection, such as general paralysis, trypanosomiasis, epidemic encephalitis, Heine-Medin's disease and the so-called enzootic encephalomyelitis of horses (Borna disease[1]).

Best studied are the cases belonging to the infectious group chiefly represented by paralytic dementia and epidemic encephalitis. These two morbid entities exhibit such typical interstitial and parenchymatous changes that they deserve to be dignified by the term encephalitis, that is to say, inflammation of the brain tissues. Being essentially alike in their histologic changes, they differ principally in the localization of the lesions which are mainly cortical in general paralysis and mesencephalic in epidemic encephalitis.

The nonsuppurative cerebral lesions caused by organic or inorganic poisons are somewhat different from the foregoing group, the difference being, in some respects, striking enough to permit a differential diagnosis under the microscope.

This contribution deals with a short review of brain changes resulting from two inorganic poisons—lead and arsenic. The changes produced by lead poisoning have been studied in three cases which were followed up clinically and confirmed by chemical postmorten examinations. The arsenical changes have been studied in photomicrographs and specimens from experimental encephalitis which Dr. A. Jacob of

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* From the pathology laboratories of Cook County Hospital, and Illinois State Psychopathic Institute.

* Presented before the first annual meeting of the Association for Research in Nervous and Mental Diseases, New York, Dec. 28-29, 1920.

[1] Joest, E.: Untersuchungen über die pathologische Histologie, Pathogenese und post mortale Diagnose der seuchenhaften Gehirn-Rückenmarksentzündung (Bornaschen Krankheit) des Pferdes, Deutsch. Ztschr. f. Nervenheilk. **42:** 293, 1911; **44:**206, 1912.

Hamburg produced in monkeys by intraspinal arsphenamin injections. He kindly placed at my disposal some of the stained sections and a great number of his photomicrographs.

Lead poisoning is usually a chronic or subacute disease process, but it seems desirable to contrast the pathologic changes occurring in epidemic encephalitis with those observed in *acute* lead poisoning. Of the three cases studied two were examples of plumbism in painters, of rather prolonged duration (about ten months), while the third was an acute lead poisoning which, because of unusual clinical phenomena, will be recorded briefly.

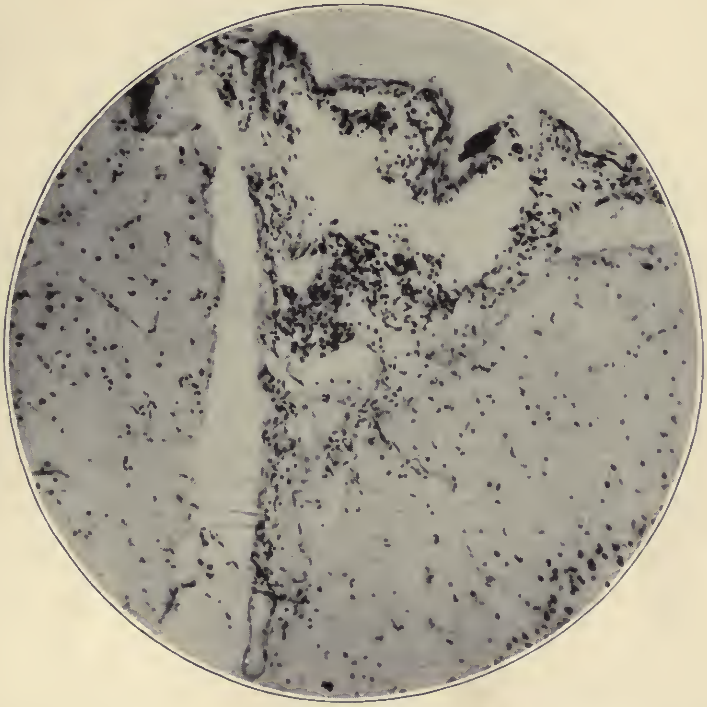

Fig. 1.—Cortex, motor area (lead encephalitis), and pia-arachnoid which is thickened and infiltrated. Some of the infiltrating cells are reproduced in Figure 6. Toluidin blue; × 120.

CLINICAL HISTORY OF AN ACUTE CASE

History.—March 29, 1920, a German housewife, 38 years old, mother of five healthy children, was admitted, in a delirious condition, to the neurologic service of Cook County Hospital. It was learned from her husband that she had always been well, had had no miscarriages, had never worked in a factory and presumably had nothing to do with lead. About five weeks before admission she was taken sick with pain in the chest and a cough. A physician prescribed medicine which she expelled by vomiting. She vomited after each meal for several days. The vomitus consisted of the food taken and did not contain blood or coffee ground masses. She was in bed for three days. Ten days prior to her entrance to the hospital she was seized with severe abdominal pain, vomiting, excessive thirst, headache and backache. For several days she complained of seeing double, and on the day before admission she became delirious.

Examination.—When admitted to the hospital the patient was disoriented and talked gibberish with but an occasional understandable phrase. The speech was tremulous, the skin pale, lemon-yellow in color. There was marked pyorrhea. and a characteristic blue line was seen along the edges of the gums. The tongue was heavily coated and the lips were covered with sordes. Nothing abnormal was discovered in the heart, lungs or abdominal organs. Paralyses, atrophies and trophic skin lesions were absent.

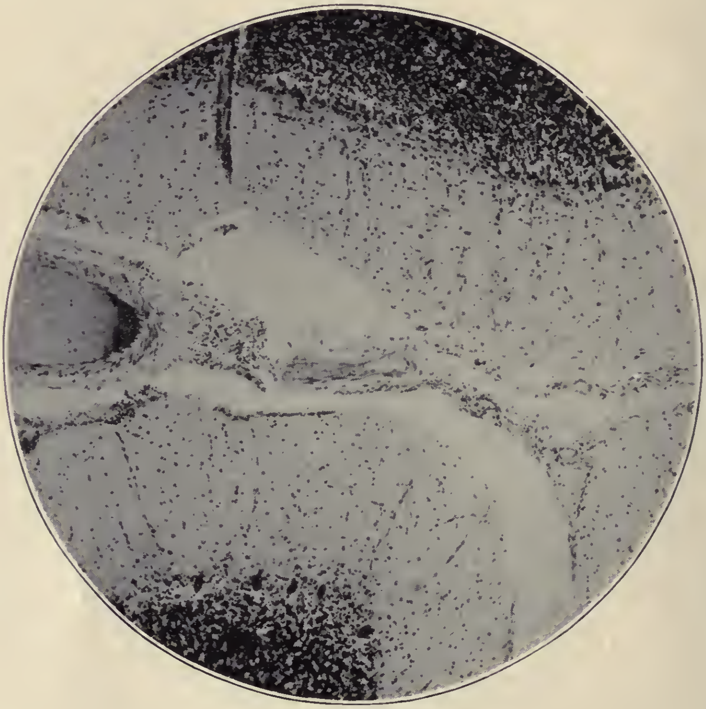

Fig. 2.—Cerebellum and its pial prolongations (lead encephalitis). The molecular layer of the cerebellum shows an abundance of tender, newly formed vessels and numerous glia cells described in the text; the pia is infiltrated with hematogenous elements shown (partly) on photomicrograph 7. Toluidin blue; × 65.

The pupils were equal, irregular and reacted sluggishly to light; there was an occasional nystagmus and a tremor around the mouth during talking. Otherwise the cranial nerves appeared normal. The tendon reflexes were normal; the abdominal reflex was absent; and the plantar reflexes were normal without Babinski or Oppenheim phenomena.

The spinal fluid was under low pressure, with 112 lymphocytes per cubic millimeter. The Lange and Wassermann tests were negative. The blood gave a negative Wassermann reaction and counts revealed 2,340,000 red cells and 17,900 white cells per cubic millimeter: Micro-, macro-poikilocytes and normoblasts were present but no basophilic red cells.

Differential count revealed: small mononuclears 4 per cent., large mononuclears 15 per cent., and polymorphonuclears 70 per cent. The urine was of dark amber color and contained traces of albumin with many granular casts and epithelial cells. The temperature, normal at the time of admission, was during the patient's four days' stay in the hospital 103 and on the day of her death 104 F.

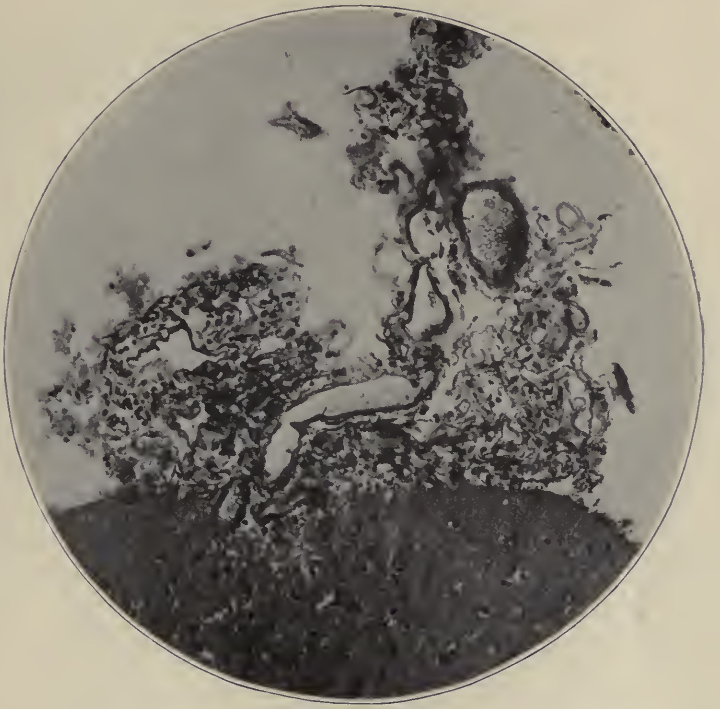

Fig. 3.—Cerebellar pia (lead encephalitis), thickened and hyperplastic with an abundance of vessels and cellular elements. Perivascular infiltrations are lacking. Combined Bielschowsky-Alzheimer-Mann stain; × 150. Compare with Figure 16.

The main macroscopic findings at necropsy, as recorded by the pathologist of the Cook County Hospital, were: marked so-called lead line of the gums (lead poisoning), marked fatty changes in the liver, icterus, acute generalized emaciation, edema of the leptomeninges, acute simple perityphlitis, hyperemia of the bowels, hyperemia of the gastric mucosa and slight edema of the lungs.

Diagnosis.—The diagnosis of lead poisoning was made during life and was based on the presence of the characteristic lead line on the gums with severe abdominal pain, vomiting and anemia. No history of exposure to lead was obtained at first, but later it was learned that "she drank some lead (by mistake?)."

Comment.— Certain features in the clinical history are unusual in lead poisoning, namely, fever, diplopia and sluggish pupils. These symptoms might raise the suspicion of a delirious type of epidemic encephalitis. The fact of lead poisoning, however, appeared to be fully substantiated and the histologic findings, to be described, are identical with those which have been described by others both for experimental and accidental plumbism. They are in no way different from those made in the other two cases. The only reason for describing in detail this case in which there might be raised the question of diagnosis is that the poisoning was acute and hence more exactly comparable with epidemic encephalitis. As will be seen from the description which follows, the microscopic changes are entirely different from those of the latter disease. The facts thus afford a striking example of the possibility of differentiating between the two forms of encephalitis by histopathologic studies.

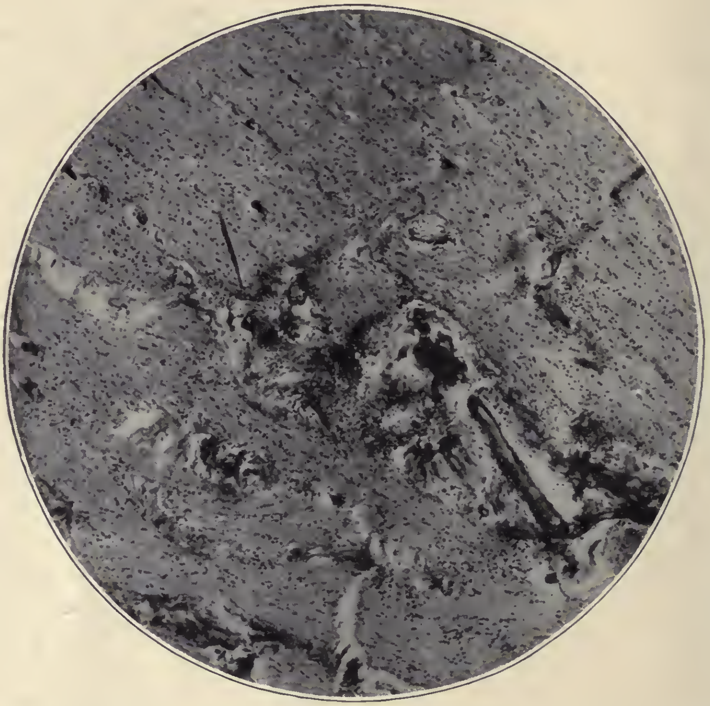

Fig. 4.—Optic chiasm (lead encephalitis). The parenchyma is infiltrated with connective tissue and vessels. Alzheimer-Mann stain; × 60.

HISTOLOGIC STUDY

The changes described herewith and the illustrations are taken from Case 3 but are identical with those from the two more chronic cases. They correspond also with the changes described as lead encephalitis by Mott[2] and Bonfiglio.[3]

1. *Pia-Arachnoid and Subarachnoid Space.*—The pia-arachnoid (Figs. 1, 2, 3 and 4) was thickened and hyperplastic. There were numerous blood vessels and the distended meshes contained many scattered cellular elements, most of them ill defined in character. Present all over the cortex, the pia-arachnoid changes were especially marked on the base of the brain and in the region of the cerebellum and optic chiasm. As seen in Figure 4, the parenchyma of the latter was invaded by large strands of hyperplastic connective tissue, derived from the pia, which consisted mainly of thickened and obliterated vessels. The latter were also quite abundant in the cortical pia and its intracerebral and intracerebellar prolongations (Fig. 3). These revealed a wealth of newly

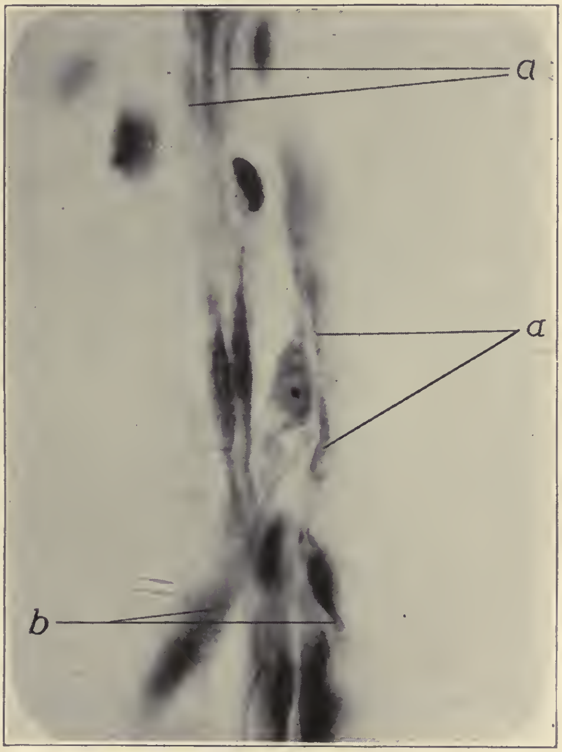

Fig. 5.—Cerebellar pia (lead encephalitis). Proliferated capillaries. Hypertrophy of the endothelial cells, their elongation, the buddings (B) are very marked. A A A, adventitial cells; the rest are endothelial cells. The adventitial spaces, especially to the right, between the adventitial cells (A) and the large endothelial cells are quite marked. Toluidin blue; ×1200.

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[2] Mott, F. W.: Examination of the Nervous System in a Case of Chronic Lead Encephalitis, Arch. Neurol. & Psychiat., London **4**:117, 1909.

[3] Bonfiglio, F.: Circa la alterazioni della cortecia cerebrale consequenti ad intossicazione sperimentale da carbonato di piombo (Encephalite productiva), Nissl-Alzheimer's Arb. **3**:359, 1909.

formed blood vessels and capillaries (Fig. 5) surrounded by proliferated fibroblasts which were frequently packed with greenish or dark granules.

The endothelial cells of the capillaries (Fig. 5) were well developed, hypertrophied, brightly stained and rich in chromatin. Frequently they showed signs of proliferation and budding. There was no perivascular infiltration though there were sometimes numerous cellular elements of various types scattered among the vessels. Many of these cells (Fig. 6) were dark, round and deeply stained and some of them on closer examination showed numerous minute granules and a pale rim of cytoplasm. Some cells were rather broken up and appeared ragged, either freely scattered in the pial meshes or gathered near the blood vessels in the form of clusters. These were evidently modified lymphocytes or plasma cells. In some places they possessed a large amount of homogeneous, metachromatically stained cytoplasm. This, in some instances. appeared granular or reticular, surrounding a pale, oval, eccentric nucleus and the cells thus very much resembled the so-called "gitter" cells. The former type, with homogeneous cytoplasm, may be classified as polyblasts, or large mononuclear lymphocytes. In addition to these three types, lymphocytes, poly-

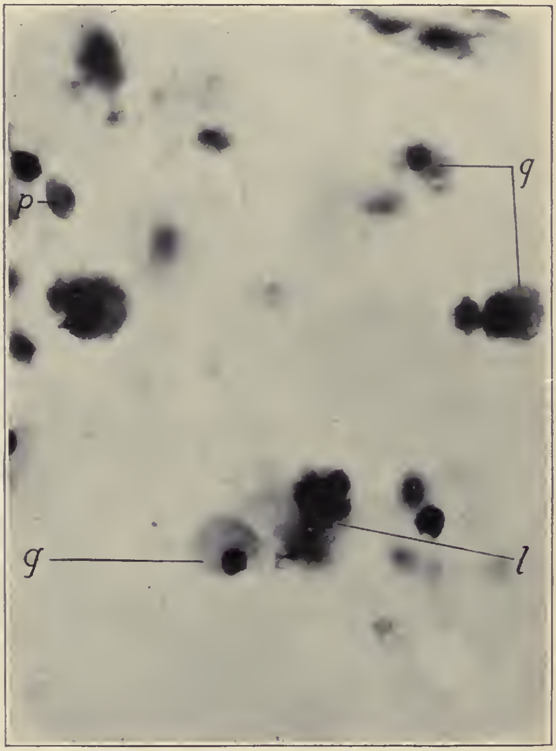

Fig. 6.—Some of the cells of the cortical subarachnoid space (lead encephalitis). The majority of these are modified lymphocytes. The large reticulated protoplasmic bodies are probably gitter cells; some are degenerated plasma cells, polyblasts and clusters of lymphocytes. P, plasma cells; G G, gitter cells; L, cluster of lymphocytes. Toluidin blue; × 1200.

blasts and gitter cells, there were a great many fibroblasts, as well as large, oval, pale bodies (Fig. 7, A) containing a few chromatin granules, located singly or in clusters. These were most probably the so-called cover arachnoid cells ("Belegzellen") usually described as mesothelial cells (Weed, Essick). The masses of cellular elements, the abundance of vessels and the distended pial meshes caused the meninges to appear greatly thickened (Fig. 3). On the base, especially around the cerebellum, the foregoing elements were very numerous, sometimes forming dense foci (Fig. 7) consisting of well preserved lymphocytes, plasma cells and many other less typical cell bodies. Such dense foci, however, were present only in the acute case and were much less in evidence in the other two. The foregoing cells did not invade the parenchyma of the cerebellum, as in epidemic encephalitis, but were strictly confined to the pia, its meshes, and the neighborhood of vessels. In the region of the optic chiasm the cellular infiltration was either entirely lacking or comparatively mild, being replaced, as noted, by an abundance of connective tissue and vessels which in some places completely obliterated the invaded parenchyma.

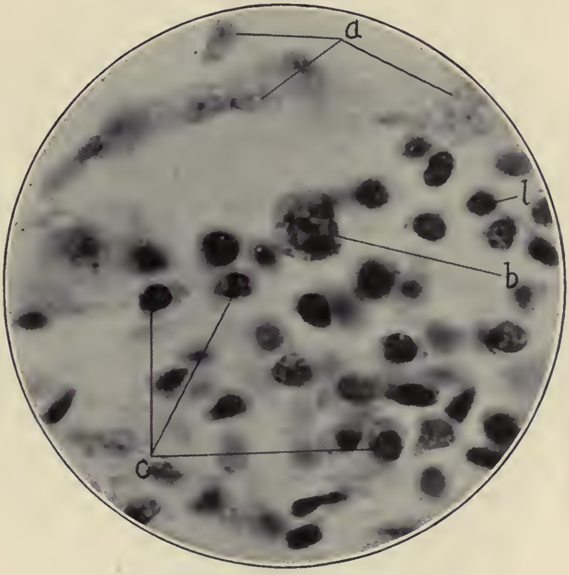

Fig. 7.—Focus of infiltration of the cerebellar subarachnoid space (lead encephalitis). A, mesothelial cells of the arachnoid, hypertrophied; B, large plasma cell; C, smaller plasma cells; L, lymphocytes. Other cells are less well defined. Toluidin blue; × 1200.

In the spinal cord (only the upper cervical portion was available) the subarachnoid space and the pia showed the presence of pigment, lymphocytes and other elements, mostly ill defined.

Summary of Pia-Arachnoid Changes: There were progressive or proliferative changes, especially pronounced on the base of the brain, around the cerebellum and in the optic chiasm. Aside from the marked proliferative or hyperplastic phenomena, there were also present in some areas, especially in the acute case, pure inflammatory changes in the form of infiltration with lymphocytes and plasma cells.

2. *Parenchymatous Changes.*—The various regions of the cerebral cortex, the basal ganglions, midbrain and cerebellum have been studied with toluidin blue, thionin, Herxheimer's scarlet red and other methods.

The architecture of the cortical layers in the various regions was well preserved and easily recognized. The subpial zone (stratum zonale) of the cerebral cortex and the molecular layer of the cerebellum (Figs. 1 and 2) were covered with abundant glia cells well supplied with cytoplasm and thin, pale, ramifying processes. Their nuclei were quite large and rich in chromatin, while the ramifying processes could in some instances be followed for a long distance, sometimes even reaching the walls of the capillaries. In the cerebral white substance (Figs. 8 and 9) the glia cells were mostly represented by nuclei, often gathered in rows along the smaller vessels and capillaries. They were always situated outside the adventitial space and frequently formed clusters. More complicated gliogenous formations, such as myeloblasts, myelophages, and the various types of gitter cells could not be found. Stained with scarlet red, the glia cells sometimes showed fat in the form of minute globules (Fig. 10). In the white cerebral substance the glia cells were numerous, and in some places the glia tissue appeared reticulated.

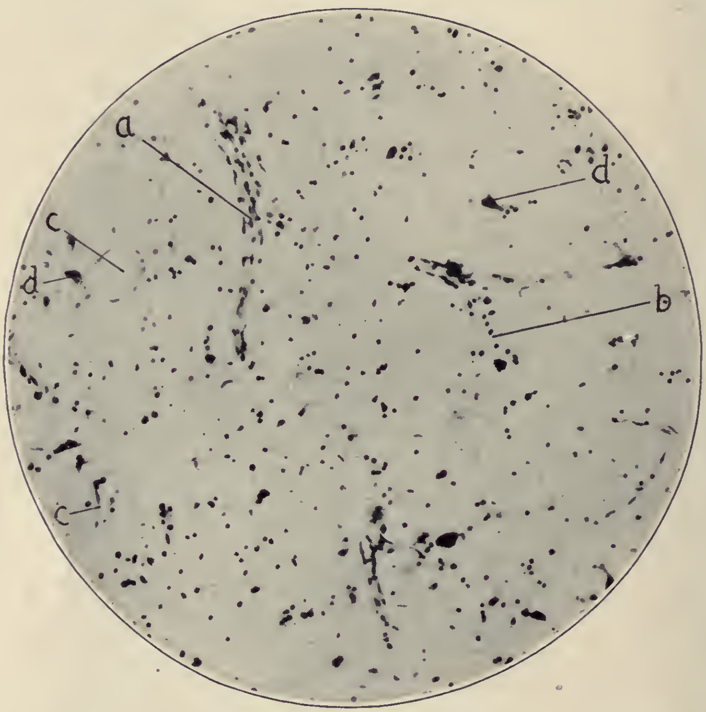

Fig. 8.—Midbrain (region of sylvian aqueduct) fom a case of lead encephalitis. Three prominent capillaries. At A, the upper portion shows two parallel capillaries; at B, a row of glia cells is well marked. The endothelial cells, even in the smallest capillary C, are prominent. D, ganglion cells. The numerous black dots are glia cells in which with the help of a hand lens protoplasm can be seen. Toluidin blue stain; × 130. Compare Figure 14.

The ganglion cells appeared normal, though their processes frequently were diffusely stained and tortuous and could be traced for a long distance. The cytoplasm was homogeneous, often broken up (chromatolysis), the nucleus eccentric with well preserved membrane and chromatin substance.

Some cells, especially those of the deeper layers, were densely surrounded and even invaded by glia cells (satellitosis and neuronophagia). In some areas, for example, the cerebellum, the ganglion (Purkinje) cells showed marked vacuolation (Fig. 11), while in the midbrain the ganglion cell changes were almost lacking. Stained with scarlet red, the ganglion cells uniformly and universally showed the presence of fat in the form of large globules within the cell body and its processes but the nucleus was left intact (Fig. 10)

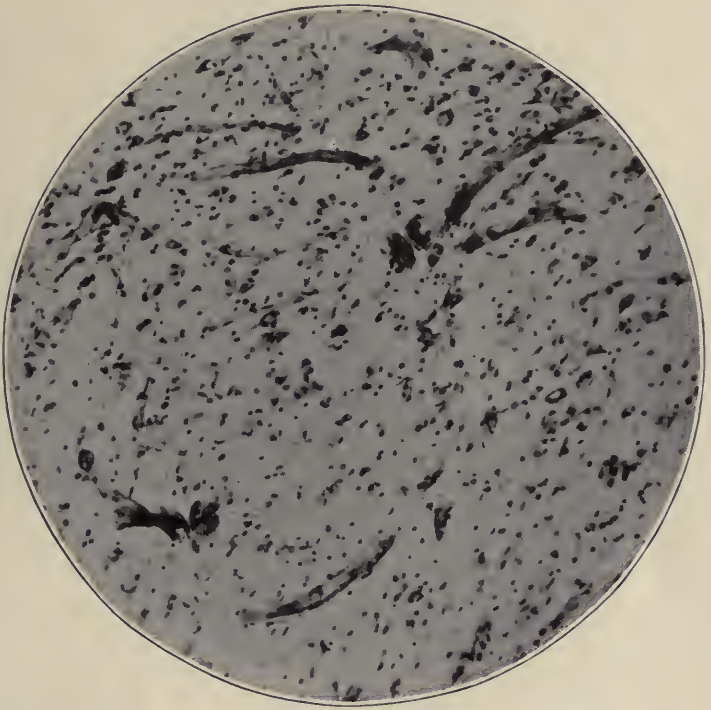

Fig. 9.—Temporal lobe (lead encephalitis). The entire field is covered with an abundance of proliferated blood vessels containing prominent endothelial and adventitial cells. The contrast with Figure 15 is marked. The abundance of glia nuclei is striking. Toluidin blue; × 130.

The nerve fibers of the brain and spinal cord (cervical region), stained with Bielschowsky or Alzheimer-Mann methods, did not appear changed; sometimes they were somewhat thickened and tortuous, especially in the subcortical area, but there were no signs of degeneration or inflammation.

Summary of Ectodermic Changes: The glia cells, like the pia-arachnoid, showed marked progressive changes; increased size of nuclei, abundance of chromatin and cytoplasmic processes and a great proliferation of glia nuclei gathered in rows or clusters. They usually contained fat and frequently invaded the ganglion cells. Most of the latter were well preserved, some showing chromatolytic changes, vacuolation and neuronophagic phenomena.

3. *Vascular Changes.*—The cortex, subcortical white substance, large ganglions, midbrain, cerebellum, choroid plexus, meninges and spinal cord (cervical region) all showed an excessive vascularization. The smallest capillaries were much in evidence, being usually engorged with blood and exhibiting unusually well stained endothelial and adventitial cells. Some capillaries were exceedingly small and narrow and contained no blood cells, often being represented by a few proliferated endothelial or adventitial cells or by offshoots, buddings or slight protrusions of a vessel wall.

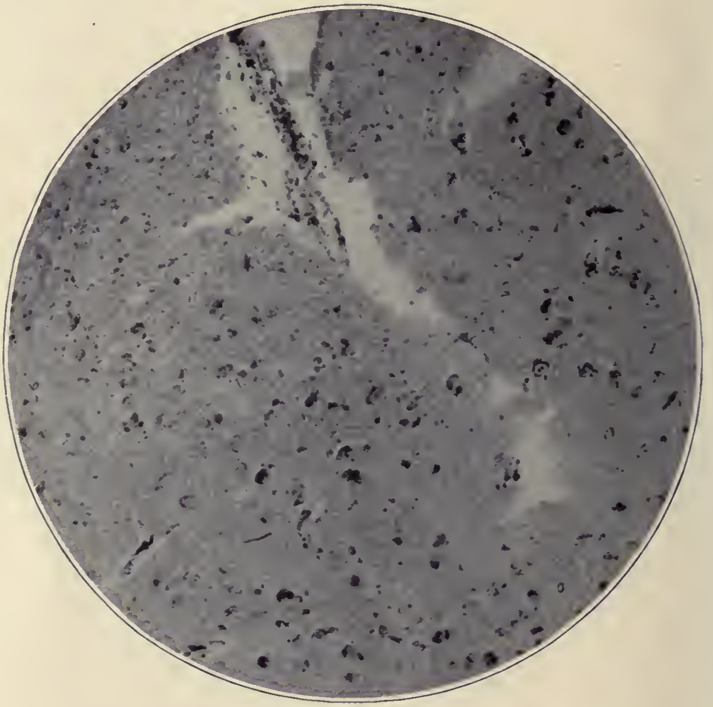

Fig. 10.—Temporal lobe. The ganglion cells, many glia cells, and especially the blood vessel (the upper part of the picture), show fat globules in the form of black spots. Herxheimer scarlet red; × 150.

The appearance of the endothelial cells, rich in chromatin, brightly stained and with cell body elongated as if thinned out, is well shown on the accompanying photomicrographs (Figs. 5, 12 and 13). The budding formations and the endothelial changes, as well as the general appearance of some newly forming capillaries, are especially well seen in Figure 5. Similar, and even more striking, new vessel formations may also be seen in abundance in the cerebral and cerebellar pial prolongations, in which they are found in every stained specimen. In fact, newly formed capillaries predominate and are much more numerous and conspicuous than fully developed preformed vessels.

In some smaller vessels and capillaries the adventitial and endothelial cells were distinctly arranged in the form of membranes separated by an adventitial space (Fig. 5) which, however, were nowhere infiltrated. The endothelial, as well as the adventitial, cells were frequently filled with greenish granules; and, as already noted, in the cerebellar pial prolongations they were densely surrounded by lymphocytes, plasma cells and other hematogenous elements.

The walls of the larger vessels were appreciably thickened, the thickening being due to proliferation of the adventitial cells and a great accumulation of fibroblasts, some of which were packed with pigment granules. The endothelial cells, as in the smaller vessels, were either increased in size with a nucleus rich in chromatin or they appeared to have proliferated and formed clusters and buddings.

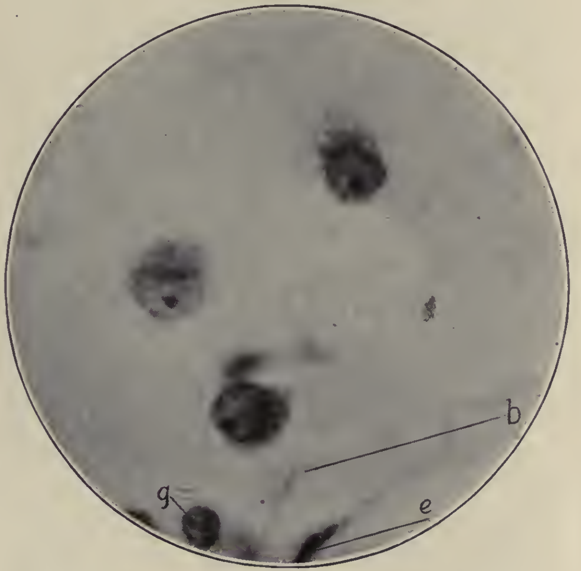

Fig. 11.—Three changed ganglion cells from the cerebellum: the nuclei are eccentric but well preserved. The cell bodies, only partly reproduced, appear pale and vacuolated. Below is a capillary with prominent endothelium (E) and budding (B); near by is a glia cell (g) well supplied with cytoplasm. Use a hand lens. Thionin; × 1200.

In the molecular layer of the cerebellum many so-called rod cells ("Stäbchenzellen") were seen. Some of them were sausage-like in shape, as if about to form a capillary, while others had visible processes emanating from a densely stained, chromatin-rich nucleus. These cells were seen only in toluidin-blue or thionin stained specimens and were absent from those stained with the Alzheimer-Mann method. In the latter they appeared as young capillaries or adventitial cells.

Like the capillaries and smaller blood vessels, the larger ones did not exhibit perivascular infiltrations. Neither was the surrounding parenchyma invaded by blood elements (lymphocytes, plasma cells), as is often the case in epidemic encephalitis and general paralysis.

Figure 8, for instance, shows the condition of the midbrain in lead encephalitis, while Figure 14 presents the corresponding area in the epidemic type. The latter is densely infiltrated, while the former exhibits only proliferating vessels.

Summary of Vascular Changes: The vessels showed marked proliferative or progressive phenomena, as evidenced by endothelial and adventitial proliferation and newly formed capillaries. Signs of perivascular infiltration, such as is seen in epidemic encephalitis, were lacking.

ARSENICAL POISONING

The findings described above for the three cases of lead poisoning are exactly similar to those found in the specimens and photographs furnished to me by Jacob, following experimental arsphenamin poisoning in monkeys. These have been described by Jacob,[4] and therefore need not be further detailed here.

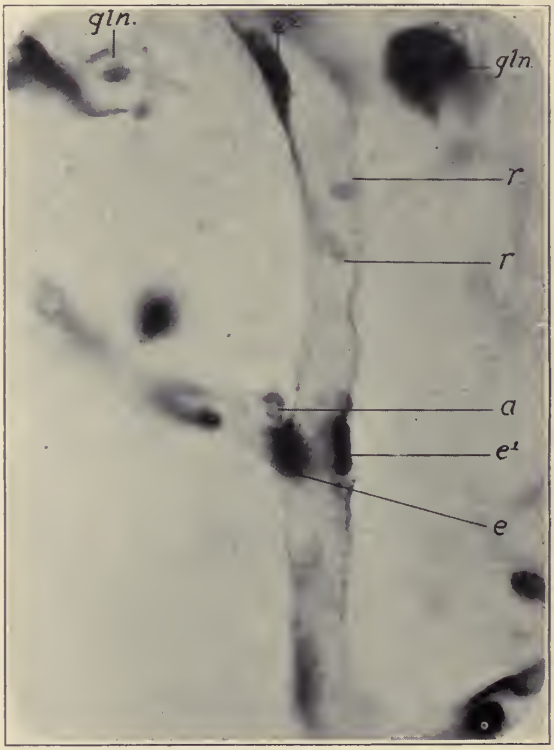

Fig. 12.—Newly formed capillaries from the cerebellar cortex E E_1E_2, endothelial cells; at E a budding formation is distinctly seen; A, adventitial cell with a cut off process (use lens); R R, red cells; Gln, ganglion cells. Toluidin blue, × 1200.

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[4] Jacob, A., Weygand, W., and Kafka, V.: Klinische und experimentelle Erfahrungen bei Salvarsaninjektionen in das Zentralnervensystem, München. med. Wchnschr. **61**:1608, 1914. Die Entzündungsfrage in Zentralnervensystem (Allgemeine Uebersicht), Jahreskurse für ärztliche Fortbildung, May, 1919.

GENERAL SUMMARY AND DISCUSSION

The changes produced in the brain tissues by inorganic poisons (lead, arsenic) are both parenchymatous and interstitial.

The former, degeneration of ganglion cells with accumulation of lipoid substances, are essentially regressive or degenerative. The interstitial changes, on the other hand, are proliferative or progressive in character (glia cell proliferation, progressive phenomena in the endothelial and adventitial cells, new formation of capillaries).

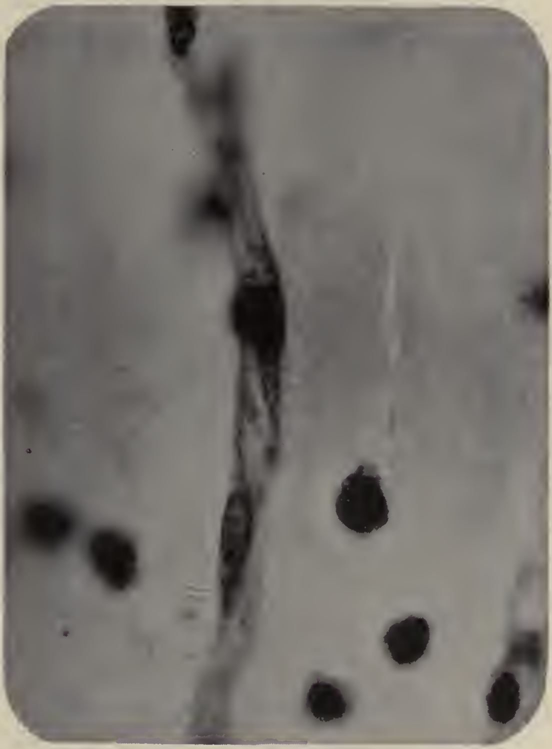

Fig. 13.—A newly formed capillary (lead encephalitis). The vessel is made up of parallel endothelial cells which contain chromatin-rich nuclei with an appreciable amount of cytoplasm at their poles. The cytoplasm is thinned out forming the capillary walls. The dark bodies outside the capillary are enlarged protoplasmic glia cells packed with chromatin. Thionin; × 1200.

While the parenchymatous changes are not peculiar or typical but resemble those to be found in any type of encephalitis, including that of the epidemic variety, the mesodermal changes of the pia and vessels are characteristic and essentially different from those seen in epidemic encephalitis. In the latter, the vascular changes are distinctly infiltrative in character and are only occasionally present in the pia (Figs. 15 and 16).

In lead encephalitis the infiltrative features are either altogether absent or are insignificant; in fact, they are to be found only in acute or peracute cases like the one in the foregoing. Instead, we find marked proliferations in the vessels as well as in the pia-arachnoid. These proliferative changes are universal but they are especially marked on the base of the brain and around the cerebellum, optic chiasm and temporal lobe. They are comparatively slight in the midbrain. The lesions in epidemic encephalitis, by contrast, always involve, though not exclusively, the midbrain.

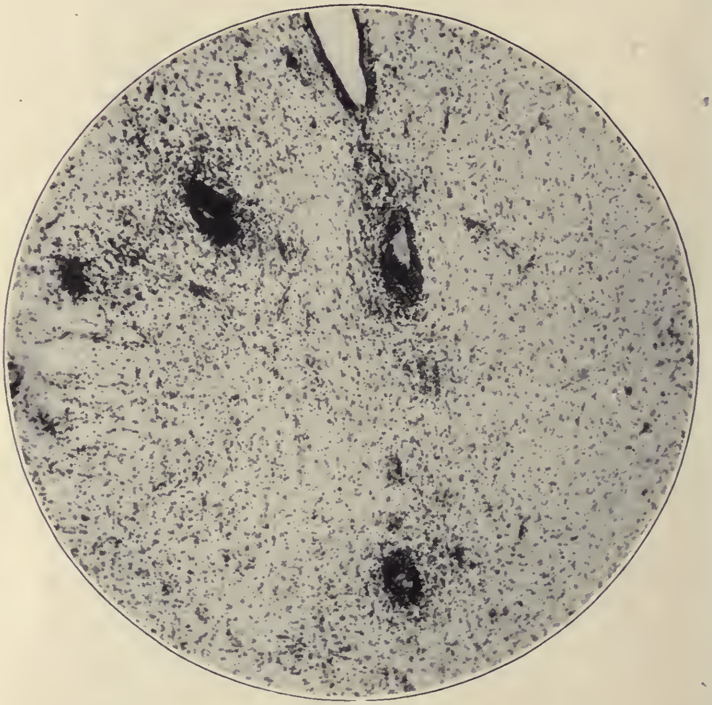

Fig. 14.—Midbrain (region of aqueduct of Sylvius) from a case of epidemic encephalitis. The vessels, as well as the parenchyma, are densely infiltrated with lymphocytes and plasma cells. Compare with Figure 8. Toluidin blue; × 65.

The prevalence of productive or proliferative phenomena in experimental lead encephalitis was also noted by Bonfiglio, who suggested for this type the descriptive designation "productive." In contradistinction to this, we might label the infectious type (to which also belong numerous other forms of nonsuppurative encephalitis caused by various infections, paralytic dementia, Heine-Medin's disease, rabies, pernicious malaria, trypanosomiasis, Borna disease) "infiltrative." It thus seems permissible to claim that vascular infiltrative phenomena are indicative of an infectious type of encephalitis, while the proliferative phenomena denote a toxic condition.

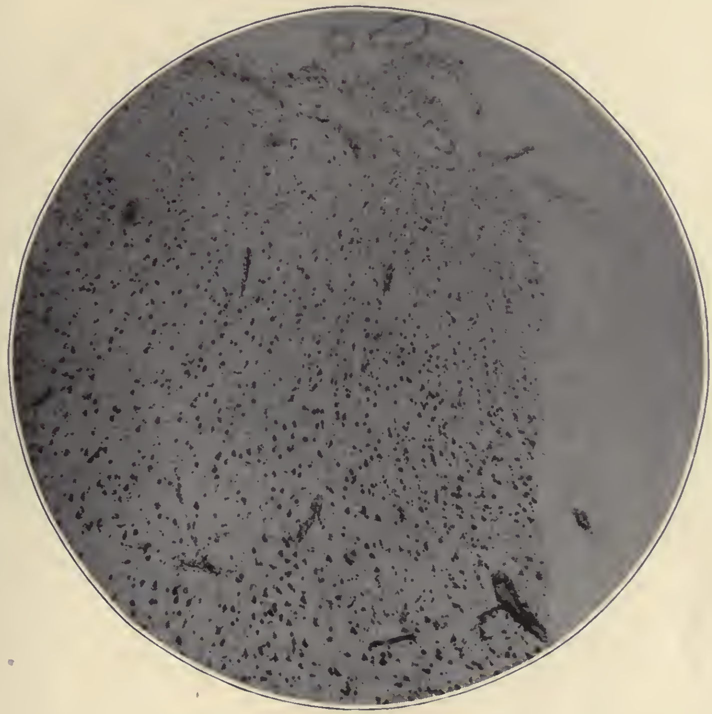

Fig. 15.—Cortex and pia from a case of epidemic encephalitis (Bassoe and Hassin). The pia-arachnoid is mildly infiltrated; the cortex shows a few infiltrated vessels, especially in the deeper layers. Compare with Figure 9. Toluidin blue; × 50.

Proliferative and infiltrative phenomena should be sought not only in the endothelial, adventitial and other vascular tissues of the brain proper, but also, and even with greater care, in the pia-arachnoid and the subarachnoid space.

In toxic encephalitis numerous, more or less ill-defined, cellular elements are mixed with mesothelial arachnoid cells, often in the form of clusters. This is not the proper place to discuss the origin and character of the various pial cellular elements, but it may be pointed out that cluster formation or proliferation of the mesothelial arachnoid cells is decidedly pathologic, for they represent a reaction against the invasion of the subarachnoid space by some irritating foreign substance. In the case of lead encephalitis this substance is some toxic product. Essick,[5] as well as Weed,[6] distinctly showed that the mesothelial cells react against irritants, such as particulate matter, and are capable of being transformed into new cell bodies resembling macrophages and under certain conditions, old age, for instance, even tumor-like masses (Weed).

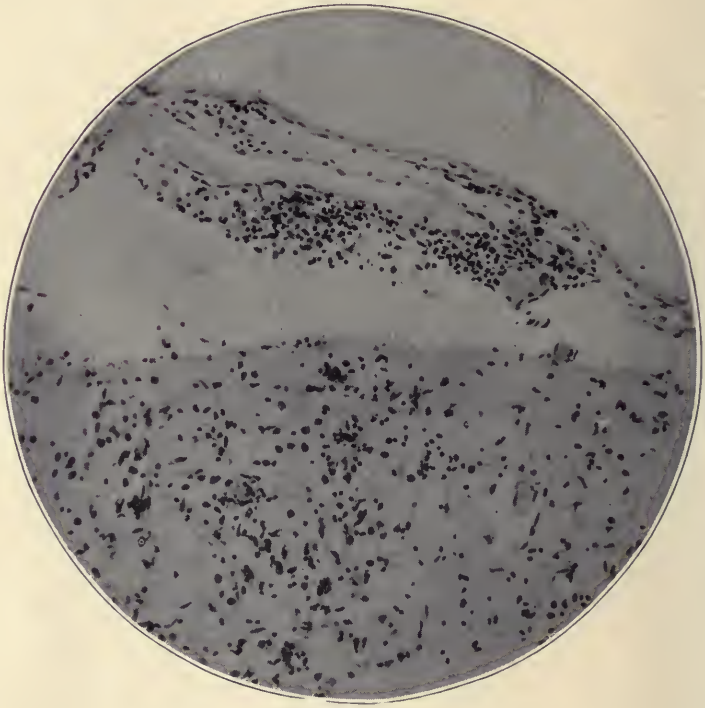

Fig. 16.—Cerebellum and pia from a case of epidemic encephalitis (Bassoe and Hassin). The pia is infiltrated, the molecular layer densely covered mostly with hematogenous elements (lymphocytes and plasma cells). Toluidin blue; × 150. Compare with Figure 3.

The condition of the subarachnoid space and its membranes may thus be considered a valuable standard for the purpose of differentiating various types of brain lesions, whether purely inflammatory, degenerative or proliferative. In fact, they may show changes even when the brain tissues proper appear quite unaffected.

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[5] Essick, Charles R.: Formation of Macrophages by the Cells Lining the Subarachnoid Cavity in Response to the Stimulus of Particulate Matter. Contributions to Embryology, No. 42, Publication 272 of the Carnegie Institution of Washington, pp. 377-388.

[6] Weed, L. H.: The Cells of the Arachnoid, Bull. Johns Hopkins Hosp. **31**:343 (Oct.) 1920.

CONCLUSIONS

1. The nonsuppurative inflammations of the brain may be divided into two large groups. One, represented by epidemic encephalitis and paralytic dementia, is of infectious origin. Pathologically, it is characterized by excessive, widespread perivascular, and sometimes also parenchymatous, infiltrations, the pia-arachnoid changes being quite mild. This type may also be defined as an infiltrative encephalitis which thus includes all possible direct infections of the brain tissue proper.

2. The second group, mainly represented by lead encephalitis, is due to intoxication and should, therefore, be termed toxic. Pathologically, it shows a great preponderance of proliferative or (as Bonfiglio puts it) "productive" phenomena in the mesodermic tissues (blood vessels, capillaries and the pia-arachnoid). This group may be termed productive encephalitis.

3. Microscopically, it is not only possible to determine whether any given case is one of a nonsuppurative encephalitis, but also whether the encephalitis is due to an infection or to an intoxication.

4. The study of the pia-archnoid and the subarachnoid space is of paramount importance in determining whether the brain is normal or pathologic, for these structures may exhibit changes even when the brain tissues proper appear normal.

STUDIES IN ASYMPTOMATIC NEUROSYPHILIS

I. A TENTATIVE CLASSIFICATION OF EARLY ASYMPTOMATIC NEUROSYPHILIS *

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BALTIMORE

Although we are as yet ignorant of the exact route followed by the *Spirochaeta pallida* in its invasion of the central nervous system, we know definitely that in many cases this invasion occurs with the generalization of the infection preceding secondary manifestations. In other words, the onset of neurosyphilis coincides in most instances with the onset of syphilis. Apparently the only exceptions are those instances in which central nervous system invasion takes place during subsequent periods of generalization of the infection, such as are believed to precede recurrent outbreaks of a secondary type; or by direct extension from an intimately associated focus of syphilitic bone or vascular disease. We may conclude that in untreated or inadequately treated cases more than one invasion of the central nervous system may occur. Ample proof of the early invasion may be found in studies of the cerebrospinal fluid in primary and early secondary syphilis.[1] In early untreated syphilis, from 30 to 50 per cent. of the fluids show more or less abnormalities, although most workers have found that only from 1 to 4 per cent. of such fluids have a positive Wassermann reaction. By means of routine lumbar puncture, therefore, it is possible in primary and early secondary syphilis to differentiate four groups of cases: one in which the fluid is normal; a second with minimal fluid abnormalities, such as slight increase in the globulin content and cell count; a third in which minimal abnormalites are associated with a positive Wassermann reaction when large amounts of fluid are used, and with a syphilitic zone gold curve or mastic "3" reaction; and a fourth group with maximal positive findings in all tests. In the first group fall the cases which presumably escape central nervous system invasion. It must be emphasized, however, that an unknown though small percentage of these may show gross abnormalities on subsequent investigation, particularly if treatment is omitted. This point is emphasized by the investigation of Steiner,[2] who with Mulzer showed that with three of twenty spinal fluids from patients with early syphilis positive inoculations in animals were obtained, although these three fluids were otherwise entirely normal. In the second group, an undetermined number are probably normal, the globulin and cell increase being irritation phenomena, comparable to the slight transient albuminuria and the appearance of a few casts so frequently observed in the urine of patients with secondary syphilis, without demonstrable kidney damage. This group, nevertheless, must be considered to contain a high percentage of cases with recent neurologic invasion, in which tissue reactions are still in abeyance or only slightly developed. The third group contains principally cases of early meningovascular neurosyphilis with possibly a small proportion of cases with incipient parenchymatous or central involvement. All cases falling in the fourth group, with all tests positive, are obviously neurosyphilitic.

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* From the Syphilis Department of the Medical Clinic, Johns Hopkins Hospital.

* Read at the Annual Meeting of the Medical and Chirurgical Faculty of Maryland, April 28, 1921.

1. Fordyce, J. A.: The Diagnostic and Prognostic Significance of Spinal Fluid Findings in Syphilis, Med. Rec. **91**:927 (June 2) 1917. Ellis, A. W. M., and Swift, H. F.: J. Exper. Med. **18**:162, 1913. Wile, U. J., and Stokes, J. H.: Involvement of the Nervous System During the Primary Stage of Syphilis, J. A. M. A. **64**:979 (March 20) 1915. McIver, Joseph: The Spinal Fluid in Primary and Secondary Syphilis, ibid. **73**:1765 (Dec. 6) 1919. Wile, U. J., and Hasley, C. K.: Involvement of Nervous System During the Primary Stage of Syphilis, ibid. **76**:8 (Jan. 1) 1921.

DIAGNOSIS AND TREATMENT OF NEUROSYPHILIS

Although the diagnosis of early asymptomatic neurosyphilis depends on information derived from spinal fluid studies only, it is interesting to note in these patients, as reported by one of us,[3] the prevalence of persistently positive blood Wassermann reactions in treated cases and the incidence of minor pupillary abnormalities—headache, lassitude, rheumatic pains and nervousness. Of a series of 642 routine spinal fluid examinations, 173 fluids were from patients presenting these manifestations (Table 1), not of themselves diagnostic of neurosyphilis, with an abnormality incidence of 28.3 per cent., while in the remaining 469 only 7.3 per cent. presented abnormalities. In this series treatment had been instituted before the lumbar puncture was done, and, as will be shown, had a modifying influence on the results.

The factor of a persistently positive blood Wassermann reaction as an indication of asymptomatic neurosyphilis may be shown by a regrouping of the results obtained in primary and secondary syphilis (Table 2). In twenty-two cases in which the blood Wassermann reaction was still positive after six doses of arsphenamin, six or 27.2

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[2] Steiner: Impfexperimente mit Spinalflüssigkeit von syphilitikern, Neurologische Centralbl. **33**:132, 1914.

[3] Moore, J. E.: The Cerebrospinal Fluid in Treated Syphilis, J. A. M. A. **76**:769 (March 19) 1921.

per cent. of the spinal fluids were abnormal. When the blood Wassermann reaction had become negative by the end of the first course of treatment, only 15.3 per cent. of the fluids were abnormal. If, after two or more courses of treatment the Wassermann reaction was still persistently positive, 38.4 per cent. of the fluids were positive; if negative, only 7.8 per cent. showed abnormal fluids. This shows, furthermore, that the prompt disappearance of a positive blood Wassermann reaction is not a guarantee against the existence of asymptomatic neurosyphilis.

Treatment as a preliminary to the study of spinal fluids in early syphilis was adopted by us a number of years ago, principally because we felt that more information could thus be gained. If the early minor abnormalities in the fluid were an expression of the general infection as an irritation phenomenon, a small amount of treatment would cause them to disappear as the general infection diminished. Under these conditions remaining abnormalities would have greater significance as indicating actual spirochetal invasion of the nervous system. That in some instances the invasion might be obscured by this amount of treatment was no objection, because, if it could be so readily obliterated, long continued treatment, our routine plan, would fully safeguard the patient. The routine treatment would not be altered because of spinal fluid abnormalities, unless their persistence indicated some alteration or addition to the therapy.

TABLE 1.—SPINAL FLUID ABNORMALITIES IN PRIMARY AND SECONDARY SYPHILIS AS COMPARED WITH BLOOD WASSERMANN REACTION AND TREATMENT

Blood Wassermann Reaction	Cerebrospinal Fluids			
	Total Number of Fluids Examined	Positive	Negative	Percentage Abnormal
After one course of treatment:				
Positive.................	22	6	16	27.2
Negative.................	39	6	33	15.3
Total...............	61	12	49	19.6
After two or more courses of treatment:				
Positive.................	13	5	8	38.4
Negative.................	102	8	94	7.8
Total...............	115	13	102	11.3

We do not advocate long delay in making the puncture nor do we believe that a single puncture should be depended on to rule out asymptomatic neurosyphilis, although negative findings in early punctures minimize the possibilities if followed by adequate treatment. In more than 100 patients in whom an early negative spinal fluid examination was obtained, reexamination of the fluid has yielded positive results in only two. In both of these an explanation for the late invasion of the nervous system was apparent. In one it probably occurred during a fresh generalization of the disease; in the second, by direct extension from syphilitic involvement of the inner cranial table. A recent communication from one of us [4] considers this point in detail.

From the foregoing it will be appreciated that the treatment of early asymptomatic neurosyphilis is so intimately dependent on the interpretation of cerebrospinal fluid findings that the two cannot be considered separately. The choice of therapeutic measures cannot be compared in importance with early recognition. We believe, however, that adequate therapy has, at this period of the invasion, a most beneficial influence on what may follow. The incidence of neurosyphilis during the period of general invasion is the strongest argument in favor of the adequate treatment of all syphilis. In addition to the data already available in the literature, a series of cases studied by us from this point of view tends to confirm this opinion.

TABLE 2.—SPINAL FLUID ABNORMALITIES IN SECONDARY SYPHILIS AS INFLUENCED BY TREATMENT

Amount of Treatment	Cerebrospinal Fluids			
	Total Number of Fluids Examined	Positive	Negative	Percentage Abnormal
None or very little	19	5	14	26.3
Six doses of arsphenamin ± mercury	42	9	33	21.4
Twelve doses of arsphenamin + mercury	37	7	30	18.9
Eighteen doses of arsphenamin + mercury	22	2	20	9.0
Twenty-four or more doses of arsphenamin + mercury	31	1	30	3.2

The influence of treatment in 151 cases of primary and secondary syphilis as indicated by changes in the cerebrospinal fluid was studied (Table 2). Nineteen patients had received little or no treatment and five, or 26.3 per cent., presented fluid abnormalities; of forty-two cases following six doses of arsphenamin with or without a course of mercury nine or 21.4 per cent. presented abnormalities in the fluid; of thirty-seven patients receiving twelve doses of arsphenamin with courses of mercury, fluid abnormalities were present in seven or 18.9 per cent.; 9 per cent. of patients in twenty-two cases following eighteen doses of arsphenamin with courses of mercury had abnormal fluids; and in thirty-one patients there were only 3.2 per cent. abnormal fluids after twenty-four or more doses of arsphenamin with courses of mercury. In this series, then, effective therapy reduced the prevalence of asymptomatic neurosyphilis from 26.3 to 3.2 per cent. If a comparison is made between the first three groups and the last two, it will be seen that after twelve or less doses of arsphenamin and courses of mercury, the spinal fluid showed abnormalities in 21.4 per cent. while after eighteen or more doses of arsphenamin plus mercury the percentage dropped to 5.7.

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[4] Moore, J. E.: The Genesis of Neurosyphilis, Arch. Dermat. & Syph., to be published.

CLASSIFICATION OF EARLY ASYMPTOMATIC SYPHILIS

Further investigations are under way but we feel that a preliminary observation on the classification of early asymptomatic neurosyphilis based on the type of spinal fluid abnormalities and the corresponding results of treatment is permissible. Our four groups may be further described as follows:

Group 1.—Normal fluids. Neurosyphilis is not definitely ruled out, but we have no means of predicting which cases will later show abnormalities, except by animal inoculation experiments. The great majority of patients in this group certainly remain free from late clinical or serologic evidence of neurosyphilis.

Group 2.—Neurologic damage minimal or questionable. The spinal fluid shows pleocytosis and increased globulin content, but negative Wassermann and colloidal gold and mastic tests. These findings may be in some cases the expression of meningeal irritation only, without definite tissue invasion. Patients showing this type of spinal fluid uniformly do well on routine treatment without the addition of intraspinal therapy. The routine may be that for patients without spinal fluid changes.

Group 3.—Tissue invasion moderate. The usual early complaint is headache. The spinal fluid shows cells from 10 to 100, usually less than 50; globulin + or + +; the Wassermann reaction is negative with small quantities of fluid, and either positive or negative with larger amounts; colloidal gold curve syphilitic or meningitic zone; mastic curve to 3, or, in some instances, paretic. In general this type of patient does well, both clinically and serologically, on routine treatment without intraspinal therapy. However, we believe that to obtain the best results the dosage of arsphenamin and the number of doses to a course should be increased over the routine used for patients without neurologic invasion, that the interval between doses should be decreased, and that the total amount of arsphenamin administered should be relatively greater and the total amount of mercury relatively less than in uncomplicated cases. In only one instance have we found it necessary to resort to intraspinal therapy in order to accomplish a serologic cure. It is probable that spinal fluid changes of this type represent future meningovascular cerebrospinal syphilis, though a minority of the patients may ultimately develop parenchymatous neurosyphilis.

Group 4.—Tissue invasion definite. Complaint may be absent, or may be that of nervousness, lassitude or neuralgic pains. If careful sensory examination is omitted, neurologic abnormalities are not detected. The spinal fluid shows from 10 to 100 cells, usually more than 50; the globulin is greater in content than in the preceding two groups, ranging from + + + to + + + +; the Wassermann reaction is positive with 0.2 c.c. or less, and the colloidal gold and mastic curves are paretic. The majority of patients in this group are not serologically cured by routine treatment, regardless of alterations in the individual dose or the total amount of arsphenamin, or the interval between doses. An occasional patient may be serologically improved, but the improvement is difficult or impossible to maintain. If, after six months' treatment, no change in the intensity of the spinal fluid findings is manifest, intraspinal treatment is an indispensable adjunct to routine treatment. Even when this plan is adopted, improvement, which may be looked for in a large percentage of the cases, is slow and treatment prolonged. In all probability, this group represents future cases of parenchymatous neurosyphilis—paresis and tabes. Indeed, we have observed the development of paresis in two patients with such spinal fluid findings discovered early in the course of syphilis.

315 Professional Building.

A COMPARATIVE STUDY OF THE SUGAR CONTENT OF THE SPINAL FLUID IN DISEASES OF THE NERVOUS SYSTEM *

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This work was originally begun in an attempt to show that the sugar content of the spinal fluid is increased in lethargic encephalitis and that this finding is an important aid in the differential diagnosis of this disease, especially in differentiating it from tuberculous meningitis in children.

Foster,[1] of Boston, has recently published a paper in which he gives a series of estimations by Folin's method. His average result is 76 mg. of sugar in 100 c.c. of spinal fluid in epidemic encephalitis. In a paper read by the author before the March, 1921, meeting of the St. Louis Neurological Society, a series of seven results was quoted from the service of Dr. Foster Kennedy of Bellevue Hospital, New York, the average being 79.3 mg. of sugar per 100 c.c. of fluid in cases of lethargic encephalitis. These estimations were all made by Folin's method, and I am indebted to Dr. Kennedy for permission to make use of these figures.

The table gives a series of sugar determinations in various neurologic conditions, from the service of Dr. Sidney I. Schwab of Barnes Hospital, St. Louis. The sugar determinations were carried out in Dr. W. Olmsted's metabolic laboratory in Barnes Hospital, and I wish to thank him for his assistance and Dr. Schwab for permission to use his cases.

These results were obtained by Shaffer's [2] method and at times also by Benedict's method for comparison. Sixteen such comparisons are shown in the table and in all cases, with one exception, the results were higher by Benedict's method. The average in our series is about 21 mg. per 100 c.c. higher reading by Benedict's method. As a rule, it is about 10 to 15 mg. per 100 c.c. higher than Shaffer's method. We have only four estimations of lethargic encephalitis in our series, and the average reading was 60 mg. per 100 c.c., by Shaffer's method.

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* From the Laboratory of Neurology, Washington University School of Medicine.

[1] Foster, Harold E.: Hyperglycorachia in Epidemic Encephalitis, J. A. M. A. **76**:1300 (May 7) 1921.

[2] Shaffer, P. A., and Hartmann, A. F.: The Iodometric Determination of Copper and Its Use in Sugar Analysis, J. Biochem. **45**: No. 2 (Jan.) 1921.

FINDINGS IN VARIOUS NEUROLOGIC CONDITIONS

Name	Date, 1921	Diagnosis	Sugar Determination, Shaffer Method	Sugar Determination, Benedict Method	Blood Wassermann Reaction	Spinal Fluid Wassermann Reaction	Pandy	Cell Count	Colloidal Gold Test
1. F. F.	May 3	Combined sclerosis	53	—	Negative	Negative	++	18	2233210000
2. H. L.	April 26	Central nervous system, syphilis	50	60	++— — —	Negative	0	2	
3. C. McC.	May 14	Central nervous system, syphilis	67	—	Negative	Negative	—	—	
4. C. R.	May 14	Central nervous system, syphilis	160 138	106	—	++	—	—	Xanthochromia
5. L. S.	May 6	Central nervous system, syphilis	42	—	++++ +++ ++++ —	++++ — ++++ —	+++	125	
6. E. B.	May 3	Central nervous system, syphilis	50	62	Negative	++++ ++++ ++++ —	++	96	0122100000
7. J. W.	Feb. 7	General paralysis	60	73	++++ — ++++ —	++++ — ++++ —	+++	18	1155543310
8. J. W.	April 23	General paralysis	53	68	++++ — — —	++++ — — —	+++	13	5554443200
9. J. W.	May 5	General paralysis	69	83	—	++++ — — —	+++	11	5554321000
10. V. F.	April 27	General paralysis	45	52	Negative	+++ — — —	++	4	0012100000
11. H. M.	May 23	General paralysis	45	—	—	++++ ++++ ++++ ++++	+++	22	5554432100
12. M. M.	May 22	General paralysis	85	101					
13. M. M.	May 23	General paralysis	112	121	Negative	++++ — ++++ —			
14. M. M.	May 23	General paralysis	53	119	....	....	+++	26	
15. M. M.	May 24	General paralysis	77	143	....	....	+++	44	
16. A. K.	May 25	Tabes	72	83	Negative	....	0	1	
17. E. T.	May 9	Tabes	69	71	++++ — — —	Negative	+++	9	
18. M. H.	April 13	Tabes	64	—	Negative	++++ — ++++ —	+++	65	0001111000
19. A. B.	Feb. 7	Tabes	54	69	—	Negative	++	37	
20. F. J.	Feb. 16	Tabes	69	—	++	Negative	+++	92	
21. J. B.	May 24	Tabes	Trace	—					
22. J. R.	Jan. 25	Lethargic encephalitis	67	—	Negative	Negative	0	4	1110000000
23. H. L.	Feb.	Lethargic encephalitis	69	—	Negative	Negative	+	36	0021100000
24. J. M.	April 13	Lethargic encephalitis	62	—	Negative	Negative	++	1	1235543210
25. J. M.	May 26	Lethargic encephalitis	43	54	Negative	Negative	+	0	
26. C. M.	April 14	Psychosis	64	—	Negative	Negative	0	0	0000000000
27. H. P.	May 9	Psych.	74	—	Negative	Negative	0	0	0000000000
28. B.	Feb. 16	Hyst.	50	—	Negative	Negative	++	4	
29. G. B.	May 19	Neuros	72	—	Negative	Negative	0	0	
30. V. T.	May 2	Hyst.	50	—	Negative	Negative			
31. W. S.	May 14	Brain tumor	82	—	+	—	-		
32. D. McC.	May 21	Ps. B. palsy	56	106	Negative	Negative	......	1	
33. R. T.	June 10	Lethargic encephalitis	64	—	Negative	Negative	0	1	
34. J. P.	June 13	Lethargic encephalitis	48	—	Negative	Negative	+	1	

These were all undoubted cases of the disease, and this unexpectedly low sugar content, determined by Shaffer's method, made us wonder whether our former results by Folin's method were not to be changed in their interpretation. Perhaps it is not sugar which is responsible for the higher value in encephalitis by Folin's method, but rather some other reducing substance or substances which may not interfere in Shaffer's method.

We have found it unnecessary to precipitate the protein before carrying out the sugar estimation in ordinary cases by Shaffer's method. In xanthochromic fluids and others with increased globulin the proteins should be precipitated as in blood sugar work. (See Case 4, C. R., in the table; 160 mg. before precipitation and 138 mg. after precipitation.) Case 31, W. S., was one of brain tumor which came to necropsy. The neoplasm, presumably a glioma, replaced part of the thalamus and lenticular nucleus of one side and was encroaching on the fourth ventricle. In Case 14, M. M., and in Case 15 the fluid was blood tinged, and the results, which are quite high, are not to be depended on; the creatinin, estimated by Benedict's method, was found to be 1.7 mg. per 100 c.c. of fluid in this case. In every case tabulated the fluid was sent to the laboratory immediately after puncture, and the sugar was estimated the same day or within twenty-four hours. We have allowed fluids to stand two or more days after puncture, and the estimation after this lapse of time was essentially the same as on immediate estimation. Most of the punctures were made about two hours after meals.[3]

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[3] In addition to the references already given, the following will be found of interest:

Dopter, C.: Sugar in Cerebrospinal Fluid in Epidemic Encephalitis, Bull. de l'Acad. de méd. **83**:203 (March 2) 1920; abstr. J. A. M. A. **74**:1545 (May 29) 1920.

Weston, P. E.: Sugar Content of the Blood and Spinal Fluid of Insane Subjects, J. Med. Res. **35**:199, 1916.

Schloss, O. M., and Schroeder, L. S.: Nature and Quantitative Determination of the Reducing Substance in Normal and Pathologic Cerebrospinal Fluid, Am. J. Dis. Child. **11**:1, 1916.

Hopkins, A. H.: The Sugar Content of the Spinal Fluid in Meningitis and Other Diseases, Am. J. M. Sc. **150**:827, 1915.

Kraus, W. M., and Pardee, I. H.: Serology of Spinal Fluid and Blood in Epidemic Encephalitis, Arch. Neurol. & Psychiat. **5**:710 (June) 1921.

EPIDEMIC ENCEPHALITIS* (LETHARGIC ENCEPHALITIS)

ITS PSYCHOTIC MANIFESTATIONS WITH A REPORT OF TWENTY-THREE CASES

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The prime purpose of this communication is to analyze the psychotic features of epidemic encephalitis as they have been observed in a group of twenty-three cases admitted to the Henry Phipps Psychiatric Clinic of the Johns Hopkins Hospital, between the spring of 1919 and the summer of 1920. It also seems worth while to report the cases in some detail and briefly to analyze them from other points of view, because they are so protean. In practically all instances these cases were referred to the clinic because of mental symptoms or behavior difficulties. It is, of course, not supposed that mental symptoms play as important a rôle in the disease picture as this group would indicate. The cases were selected for us, as it were, from the larger group which, for the most part, was admitted to the general hospital. On the other hand, this series does not constitute a small proportion of the general hospital group. Not more than 100 cases have been seen in the entire hospital, and in the psychiatric dispensary a number of cases were seen which could not be admitted to the hospital.

A study of the psychotic data has made diagnosis possible in some patients who presented few neurologic signs, and the mental picture has been found to offer valuable confirmatory evidence in doubtful cases with neurologic findings. Several characteristic symptoms have been made out as well as certain general reaction types. While the reaction types are not distinctive and decisive, they are often suggestive and helpful.

Nine syndromes are presented in the order of their diagnostic importance.

SYNDROMES IN EPIDEMIC ENCEPHALITIS

1 *Great Push of Talk Without Distortion of Stream of Talk and Without Mood Alteration.*—This was first noted in Case 14. When given the slightest opportunity, the patient poured forth a minute, detailed description of all of his thoughts and feelings. At first his talk was concerned with the material of his delirium and later with

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* From The Henry Phipps Psychiatric Clinic, Johns Hopkins Hospital.

discussion of the disease, its symptomatology, treatment, etc. The only way of stopping his talking was to keep every one out of earshot. After five months of the disease, this was the only symptom persisting. In Case 10, this was the only abnormality giving a clue to the diagnosis. The mind of the second patient was quite clear but he seemed to be unable to control the incessant flow of words. The manic type of association—flight of ideas, distractibility, etc., has not been present, nor does the mood show alterations to account for the talk. The patients are not distractible; it is difficult to divert the topic of conversation to other channels. Increase of general activity has been absent. Apparently all inhibitions to silence are off; the flood gates are down and some force seems to drive from behind. This symptom was present in Cases 5, 9, 10, 11, 12, 14, 20 and 21. Mention is made of this excess of talk by two other observers, Hall [1] and House.[2]

2. *Euphoria, Jocularity, Feeling of Well Being.*—This has been one of the most striking symptoms and has been noted by Wilson [3] and House.[2] The latter regards it as one of the most characteristic symptoms. Case 1: The patient said that she "felt like a new-born babe." The patient in Case 2 seemed quite jovial, said she "felt happy" and passed witty remarks about other patients, although complaining constantly about her condition. The patient in Case 3 said she "felt all right" although she asked constantly for medicine and was very restless and uneasy. The patient in Case 5 said he felt "very well" and was cheerful. The patients in Cases 6, 9, 10 and 11 described the mood as "good." The patient in Case 13 joked in a knowing way when his illness was mentioned. The patients in Cases 14 and 21 both "felt fine" though racked with severe pain. The patient in Case 20 was quite talkative and happy during the first week of the disease.

This sense of well-being reminds one in some respects of the euphoria of the paretic, but is less empty and vacuous.

3. *Alertness and Mental Clearness Immediately on Being Aroused.*—This is evidenced when the patient is once aroused, in spite of stupor and somnolence.

When the disease first began to be seen it was amazing how the patient dropped off to sleep during an examination. The clearness and alertness immediately on being aroused is equally surprising. One would not expect as much wide-awakeness in a normal person aroused from sleep. It is probably true that the somnolence appears deeper than it really is because of the frequent ptosis. The phenomenon has been observed in Cases 1, 2, 6, 7, 9, 15 and 23, which comprise about one half of all cases showing somnolence. Attention has been directed to this finding by Economo [4] and Wilson.[3]

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[1] Hall, A. J.: Epidemic Encephalitis, Brit. M. J. **2**:461, 1918.

[2] House, W.: Epidemic (Lethargic) Encephalitis. Clinical Review of Cases in the Pacific Northwest, J. A. M. A. **74**:372 (Feb. 7) 1920.

[3] Wilson, S. A. K.: Epidemic Encephalitis, Lancet **2**:7, 1918.

4. *Delirium.*—Delirium has been noted in fourteen, possibly in fifteen, of our cases. This term means any psychosis in which there is a dreamy state with clouding of consciousness, disorientation and association in which drifting and groping are present. Following Hoch's [5] classification of the deliria as organic and psychogenic, it was found that practically all of our cases fell into the organic group. The content of the delirious utterances concerned almost exclusively topics which did not involve the personality of the patient in any of the instinctive realms. Occupation was the most common type seen (Cases 1, 5, 6, 9, 13, 14, 19 and 21). This has been the rule in cases reported by others. In three of our cases—14, 15 and 16, the delirium was accompanied by fear. In only one of these cases (14) was there any possibility of alcoholism. The only other case reported with fear, is one of Wilson's,[3] and it is doubtful whether the diagnosis of encephalitis was correct. Hallucinosis was present in seven cases, and it usually dealt with casual material—occupation objects, elevators, etc. In one case the patient saw animals with fear response.

5. *Stupor States.*—Five of our patients showed stupor at the beginning of the disease. These stupor reactions have been mistaken for schizophrenic reactions, but it seems to us that usually the differentiation is not difficult. The schizophrenic patient makes use of voluntary motor mechanisms and rarely is there the incoordinate tremor and tonic rigidity seen in the encephalitic. In addition, the stupor does not arise in a schizophrenic setting. Further, the clearness of the catatonic stupor is absent.

6. *Behavior Oddities.*—Impulsive or unusual acts totally out of keeping with the rest of the activity of the patient have been seen in seven cases. This is as one would expect in psychosis of organic basis. The impulsiveness of the paretic and of the arteriosclerotic patient is well known. Frequently, however, the oddity has been so unusual, unmotivated, and in some cases so silly, that it has been significant. For instance, the patient in Case 3 tried to commit suicide by hanging herself with a shoestring and without any real desire to kill herself. The patient in Case 1 got out of bed suddenly, dashed down the long corridor and, when brought back, said "Why do I do things like this?" She was not frightened. The act seemed quite unmotivated. Case 2:

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[4] Von Economo, C.: Die Encephalitis Lethargica, Leipzig and Vienna, 1918.

[5] Hoch, August: The Problem of the Toxic-Infectious Psychoses, New York State M. J. **13**: (Feb.) 1913.

The patient bathed practically the entire day, wrote an extraordinary number of letters, and took huge doses of cathartics. Case 11: The patient talked in an odd, stilted way, and in a high-flown, silly manner. In the midst of a formal conversation he would swear violently without any anger. Case 13: Although the patient had been in bed for weeks and was very somnolent most of the time, he would get up suddenly, go to his wardrobe, pack his suit-case and announce that he was leaving. Case 15: This patient attempted suicide on two occasions by walking out of the third story window. Case 16: This patient had temper tantrums without precipitating causes and totally out of keeping with her former disposition. Case 17: The patient would get out of bed and wander about looking for medicine. .She was not delirious and knew well that she was in isolation.

7. *Depression.*—This symptom has been observed by us in seven cases. In cases 2, 3 and 5, reported by Barker,[6] it was the most prominent symptom present. Flexner [7] and others [8] have called attention to this affective change. In our Cases 6, 7 and 21, it was transitory; but in Cases 15, 16, 18 and 20 it was pronounced and prolonged, dominating the clinical picture. Suicidal attempts have been frequent, and at least two patients were brought to the hospital for this reason. In Case 15, there was the formal alteration of thinking and activity which characterizes the manic depressive patient—that is, slowness of thinking and talking, especially in the morning. Case 17: This patient showed a great deal of puzzling bewilderment and fear, as well as depression. In Cases 18 and 20, there was no slowing.

8. *Emotional Instability and Irritability.*—These symptoms have been frequent, especially the latter. Tantrums and outbursts of anger have occurred without obvious cause. The lability and overflow of emotional response have suggested a pseudobulbar syndrome in Cases 1, 5, 8 and 9.

9. *Memory Defect.*—It was rather surprising to find so little evidence of real, permanent memory defect in our cases, since we are dealing with a disease in which there is demonstrable alteration of brain substance. In all four cases (8, 11, 13, 22) in which memory defect was demonstrated (of course the acute deliria are excluded), the prime difficulty was in recent and immediate (retention) memory. The

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[6] Barker, L. F.; Cross, E. S., and Irwin, S. W.: Epidemic Acute and Subacute Nonsuppurative Inflammations of the Nervous System Prevalent in the United States in 1918-1919: Encephalitis; Encephalo-Myelitis; Polyneuritis; Meningo-Encephalo-Myeloneuritis, Am. J. Med. Sc. **159**:157 and 337, 1920.

[7] Flexner, S.: Lethargic Encephalitis, J. A. M. A. **74**:865 (March 27) 1920.

[8] Encephalitis Lethargica: A New Disease? J. A. M. A. **72**:414 (Feb. 8) 1919; also footnote 4.

patient in Case 8, in addition, confabulated freely. With an alcoholic history, this case might easily have passed as a Korsakoff syndrome. In fact, there is a similarity here to the alcoholic deliria which become chronic and pass into the Korsakoff psychosis. All four cases showed a preliminary period of delirium, with recent memory and retention difficulty. This type of memory difficulty, with a tendency to confabulate, is, according to Bonhoeffer, the characteristic feature of alcoholic delirium. It was pointed out in the foregoing that the patients can be easily aroused to take in their environment. This is also characteristic of the alcoholic. Fear appears in several of our cases. The protracted reductions of epidemic encephalitis have, then, several points in common with the chronic alcoholic deliria.

REACTION TYPES [9]

In our twenty-three cases, four reaction types can be made out: (1) depressive, (2) psychoneurotic-like, (3) delirious, and (4) organic. While these reactions have not been clear-cut and decisive, and all have some organic coloring, they present sufficiently clear reaction tendencies to deserve grouping, especially as the psychotic features may present difficult diagnostic problems.

The inquiry arises here, as in the psychoses of parenchymatous syphilis, "Why should one patient react with manic excitement, another with depression, and a third with a classical paretic picture?" Neuropathology offers no explanation. We believe that with diffuse organic change, the personality or constitution of the patient is apt to rupture at its point of greatest vulnerability. A personality or constitution able to maintain an adequate adjustment with the proper repression or sublimation of unsocial trends or of strong instinctive drives, breaks along these lines when the automaticity of adjustment is interfered with. Some of our cases support this notion. Case 15: This patient, petted and spoiled as a child, with a violent temper, and later humored by her husband, with a tremendous sex urge which her husband was unable to satisfy, reacted with temper tantrums, and openly made sex advances to her physicians. In addition she showed tremendous desire to punish and fondle her nurses and repeatedly asked her physicians to punish her for her bad behavior. Case 16: The patient, caught by the disease in the midst of a difficult personal situation (refusal of her teachers to allow her to attend a "hop," and the knowledge that her parents were unable to allow her to return to the same expensive school the following year), reacted with temper tantrums, depression and attempts at suicide.

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[9] Under the discussion of each reaction type a short psychiatric summary of the cases of that reaction are included. The detailed abstracts of the cases will be found at the conclusion of the paper.

Case 18: A man of worrying disposition, became depressed and attempted suicide when his health appeared to be gone. Case 4: This patient, who had a history of years of hypochondriasis, reacted with a "chronic invalid" picture.

1. *Depressive Reaction Type.*—This type of reaction was observed in seven cases but in only two were there formal thinking and activity alterations, that is, slowness of the manic-depressive reactions. The other cases resembled the reactive and psychoneurotic depressions. The depression was usually combined with delirium.

Case 6: Insomnia was observed at onset; later drowsiness and stupor. The patient felt sad and wished to die. He was alert when aroused; his spirits were good, although he was sad about being away from home. There were some date discrepancies but with spontaneous correction. He recovered in three weeks. (Midbrain, rubro-striate, bulbar, pyramidal involvement).

Case 15: The patient was a petted, spoiled child, violent tempered and highly sexed. The onset was acute, accompanied by somnolence; after three weeks, there was an onset of irritable nagging depression with two suicidal attempts. There were subjective loss of memory and confusion; people were talking about her. During the course of the psychosis she showed great sex tension, with sadistic and masochistic trends; she made frequent suicidal attempts. She recovered after eight months. (Midbrain).

Case 16: Onset occurred during a period of disappointment, with depression, insomnia, emotional tantrums and suicidal attempts. During the course of the disease she was agitated, restless, suicidal. There was subjective inability to think and understand; she was puzzled and fearful. She recovered in two and one-half months. (Midbrain and bilateral pyramidal signs).

Case 18: Drowsiness and stupor were present one week after onset. The patient had nocturnal delirium for three weeks, then became restless, sleepless, weak, excitable, exacting and depressed. She worried over financial difficulties and made two suicidal attempts. (Midbrain; cerebellar).

2. *Psychoneurotic-Like Reactions.*—Several of our patients were admitted to the hospital with a diagnosis of hysteria or functional disorder. Case 3: This patient took to her bed after a severe fright and was easily startled. She would scream in the middle of the night as if in terror, and appeared dull and out of contact when attempts were made to talk to her. (Midbrain, bulbar, rubrostriate and pyramidal involvement). Case 4: The patient became increasingly hypochondriacal during the course of her illness. She always complained of somatic symptoms and her organic signs were discounted by her family

physician. (Cerebellar?) Case 10: The patient had always been nervous, emotionally unstable and hypochondriacal. The encephalitic process increased the same symptoms. Case 17: This patient complained constantly of gastro-intestinal difficulty and headache. All his symptoms were easily removed by suggestion. Any kind of capsule gave him a comfortable day. (Cortical; midbrain; rubrostriate). Case 23: The patient complained of indescribable pain all over the body, constipation, pain in the stomach, and a host of other symptoms. Just how many of these complaints were due to organic changes seems impossible to say. Our attitude has been to treat them as psychoneurotic because this opened the door to the main therapeutic aid more than did the more organic hypothesis.

3. *The Delirious Reaction Type.*—This type has already been discussed under symptomatology.

Case 1: Emotional instability and sleeplessness were present; three days later, stupor and delirium; (disoriented, saw animals on the wall, behavior oddities). There was a feeling of well being; alertness was shown when aroused. (Midbrain; bulbar involvement).

Case 2: After three months of illness the patient became irritable, discontented, restless and antagonistic. She took large doses of cathartics, bathed excessively, wrote an extraordinary number of letters, and later said she was a queen and that a drinking glass was worth a million dollars. Still later, she was stuporous but alert when aroused; also delirious, talking about trips to the mountains and imaginary conversations with negroes. She complained about the treatment she received, but was cheerful and jovial. There was a gradual recovery after five months. (Midbrain involvement).

Case 7: Onset occurred with fatigability, weakness and periods of drowsiness. Later, he had a delusion in which he thought he was talking to his dead wife and sister; he had feelings of unreality. Delirium was combined with depression. The delirium became more profound with the fear state; he thought that he was being killed and that his food was poisoned, etc. Delirium deepened into stupor and death occurred, due to bronchopneumonia. (Bulbar).

Case 9: Occupation delirium was present from the onset. Later, the patient was somnolent but was easily aroused. There were periods of excitement during the delirium; the mood was "good." Recovery occurred after one month. (Midbrain; rubrostriate).

Case 12: The onset was accompanied by weakness, restlessness and pain all over the body. There was drifting talk with partial disorientation; the mood was "all right." Attention could not be held on arousal. Delirium was more pronounced with excitement and overactivity. Death occurred one month after onset. (Midbrain; cortical).

Case 14: The onset was acute with restlessness, tension and fear. He thought he was to be shot. He had occupation delirium with drifting associations, but he was oriented. There was an incessant flow of utterances. There was persistence of fear delirium with push of talk for several weeks; then subsidence of delirium occurred with residual of irritability and constant, incessant talking. Volubility was present after one year. (Midbrain).

Case 19: Onset occurred with sleeplessness and occupation delirium and irritability. Gradual recovery occurred after two months, with residual of alteration of the sleep curve and pains in the chest. Complete recovery took place after eight months. (Rubrostriate; bulbar; midbrain).

Case 20: The patient was talkative and happy during the first week; blue, inactive and quiet during the second week; he became sleepless. Depression then deepened; he complained of pain around the heart and smothering sensations. The latter resembled anxiety states and the patient felt as though he were dying and called for help. Then followed a period of muttering, fluctuating delirium. With the onset of profound rigidity the patient became mute and stuporous. After a period of many months he began to show that he understood and would answer by closing his eyelids. He gradually began to speak and gave evidence of clearness, but developed several paranoid trends—he thought that the intern had given him syphilis, cystitis, etc. He still shows profound rigidity after ten months. (Midbrain; rubrostriate; bulbar).

Case 21: Four days after the onset he complained of feeling nervous. He became restless and talked incessantly. He did not sleep from the onset until death seventeen days later. He moved about constantly as if in great pain, and his talk concerned his occupation. He was partially disoriented, and in spite of great pain said he was in good spirits. Attention drifted easily. (Midbrain; bulbar).

4. *Organic Reaction Type.*—This has been characterized by the presence of memory defect, mainly for recent events and retention. Good humor, a sense of well being and a tendency to confabulate were present.

Case 8: The patient had influenza February, 1919, followed by weakness and "nervousness." June, 1919, he had delirium; he improved, but had to give up work on account of mistakes. He felt confused and then became drowsy and delirious again. He began to confabulate; he wept and laughed with an overflow reaction. Reduction in memory and retention were present. Association assets were poor (Korsakoff syndrome). (Rubrostriate; midbrain; cerebellar; pyramidal involvement).

Case 11: Onset occurred with weakness and fatigue; he soon became irritable and for two weeks was speechless after a temper tantrum. His behavior was "peculiar"; he swore without anger or cause; his talk was stilted; his mind appeared to be clear but he did not know persons or places. Memory for recent and remote events was poor. There was a general reduction of association material. Speech and writing were careless. Symptoms persisted until death after one year. (Optic neuritis and pyramidal signs).

Case 13: Onset was gradual with loss of interest, fatigability and spells of unconsciousness. Memory showed gradual deterioration over a year. On admission to the hospital he was drowsy. He fell asleep during the examination but was easily aroused. He was disoriented; his mood was "good"; he wandered about the ward looking for imaginary persons and places; he thought detectives were hunting him; he was jocose and covered his mistakes by joking. There was a general reduction of association assets; he showed no insight. There was no change after eighteen months. (Bulbar; rubrostriate; cerebellar).

TABLE 1.—Psychotic Manifestations

Case Number	Delirium	Feeling of Well Being	Alertness and Clearness When Aroused from Stupor	Over-talkativeness	Emotional Instability	Depression	Behavior Oddities	Memory Defect
1	+	+	+	0	+	0	+	0
2	+	+	+	0	0	0	+	0
3	0	+	0	0	0	0	+	0
4	0	0	Not somnolent	0	0	0	0	0
5	+	0	0	+	+	0	0	0
6	0	0	+	0	0	+	0	+ (?)
7	+	0	+	0	0	+	+	0
8	+	+	0	0	+	0	0	+
9	+	+	+	+	0	0	0	0
10	0	+	Not somnolent	+	0	0	0	0
11	+	+	Not somnolent	+ (?)	+	0	+	+
12	+	+	Not somnolent	+	0	0	0	0
13	+	+	0	0	0	0	+	+
14	+	+	Not somnolent	+	0	0	0	0
15	0	0	+	0	0	+	+	0 (?)
16	0	0	Unknown	0	0	+	+	0
17	0	0	0	0	0	0	0	0
18	+	0	0	0	0	+	0	0
19	+	0	Not somnolent	0	0	0	0	0
20	0	0	Not somnolent	+	0	+	0	0
21	+	0	Not somnolent	+	0	0	0	0
22	+	0	0	0	0	0	0	+
23	(?)	+	+	0	+ (?)	+	+	+

Case 22: July, 1919, the patient showed a let-down in his activity, endeavor and good spirits. January 1, 1920, he began to sleep poorly and was restless. January 14, he became drowsy and listless. During February he had "mild convulsions." At night he was slightly delirious. Toward the end of February he began to improve but continued to feel weak and fatigable, and memory was poor, especially for recent events. There was no change in his condition during treatment. (Cortical; midbrain).

The results in the foregoing cases are summarized in Table 1.

SUMMARY OF PSYCHOTIC MANIFESTATIONS

1. Nine symptoms have proved to be of frequent occurrence: (*a*) push of talk without distortion, (*b*) surprising alertness on arousal from stupor, (*c*) feeling of well-being and happiness, (*d*) behavior oddities out of keeping with general activity or mood, (*e*) emotional instability and irritability, (*f*) organic type of delirium at times accompanied by fear, (*g*) stupor states, (*h*) depression frequently the dominant mood, and (*i*) characteristic memory defect.

2. Four reaction types have been made out: (*a*) depressive, (*b*) psychoneurotic-like, (*c*) delirious, and (*d*) organic.

ANALYSIS OF CLINICAL COURSE AND SYMPTOMATOLOGY (SUMMARIZED IN TABLE 2)

1. *Onset and Signs of Infection.*—Onset: The onset was acute in thirteen cases, subacute in six and gradual in four. The most common symptoms were dizziness, pain in the head, neck, body and extremities (probably root pain), headache, fatigability, excitability, restlessness, insomnia and acute respiratory disease symptoms. Nausea and vomiting, depression, staggering gait, muscle twitchings, etc., were occasional symptoms. Fever at the onset was mentioned in only three cases.

TABLE 2.—SYMPTOMATOLOGY

Case Number	Onset	Lethargy and Somnolence	Asthenia	Fever	Diplopia	Headache	Insomnia	Noise Hypersensitiveness	Pain
1	Acute	+	+	+	+	+	+	+	0
2	Acute	+	+	+ (?)	+	+	+	+	+
3	Subacute	+	0	+	+	+	+	0	0
4	Acute	0	?	+	0	+	+	0	0
5	Acute	+	+	+	+	+	0	0	0
6	Gradual	+	+	+	0	+	0	0	+
7	Gradual	+	+	+	0	+	+	0	+
8	Gradual	+	+	+	0	+	+	0	0
9	Acute	+	+	+	+	+	0	0	+
10	Acute	0	+	+	0	+	+	+	0
11	Subacute	0	+	+	0	0	0	0	0
12	Acute	0	+	+	+	0	0	0	+
13	Gradual	+	+	0	0	+	0	0	+
14	Subacute	0	+	+	+	0	+	+	0
15	Acute	+	+	+	+	0	+	0	0
16	Acute	+	0	+	+	0	+	0	0
17	Acute	0	+	+	+	+	+	0	0
18	Acute	+	+	+	+	+	+	+	0
19	Subacute	0	0	+	0	+	+	0	+
20	Subacute	0	+	+	+	0	+	0	+
21	Acute	0	0	+	+	+	+	0	+
22	Subacute	+	+	0	+	0	+	0	0
23	Acute	+	+	+	+	+	+	0	+

Fever has been present in all but three of our cases, and the history is doubtful in one of these three. It has usually been only slight or moderate (100-102.5 F.) except in fatal cases. There appears to be no correlation between the degree of pyrexia and the neurologic or psychotic symptoms. The fever has been irregular in all of our cases.

2. *Brain Infection.*—Headache was noted in seventeen cases. In some it was exceedingly severe and persistent.

3. *Disorder of Brain Stem.*—Diplopia was present in fifteen of the twenty-three cases. It was usually transient but persisted in a few for as long as one month.

4. *Involvement of Basal Ganglions.*—Lethargy or somnolence was present at some time in the course of the disease in fourteen cases; at onset in three, within the first week in five, and within three weeks in five cases. Somnolence was present for from a few days to many months, and frequently was intermittent. In two, it appeared late in the disease.

Insomnia was present in seventeen cases. This bears out the contention that the disease is incorrectly named. We have chosen to accept the suggestion of English observers that the disease be called epidemic encephalitis and the type specified when the etiologic factor is known. In many cases the insomnia was intense and prolonged. In some cases, with the coming of night the patient became wide awake and active. In one case (22), it was impossible to induce sleep.

5. *Cerebellar Involvement.*—Asthenia, more or less marked, has been a constant finding (eighteen cases). It may appear at any stage of the disease and usually persists for a long period after the disappearance of other symptoms.

6. *Middle Hindbrain Segment Involvement.*—Hypersensitiveness to noises was observed in five cases and was quite striking. A loud or shrill noise frequently caused intense suffering, and at times outbursts of anger and irritability.

7. *Spinal Root or Spinothalamic Mechanism Involvement.*—Pain: The pain was of various types. It ranged from general aching to severe lightning pains. It has been suggested that the lightning pains were due to inflammation of the spinal roots, although the absence of herpes casts doubt on this. It is possible that the spinothalamic mechanism was disturbed.

PROGNOSIS

Of the twenty-three patients in our series, five died. Three of them ran a chronic course (many months), and two died after an acute course (two weeks). Twelve have made practically complete recovery. Of the remaining six, two are in a critical condition, and the other four are on the road to recovery.

NEUROLOGIC FINDINGS

There was third nerve involvement in seventeen cases. Ten of these showed involvement of the extra-ocular muscles. Ptosis was

TABLE 3.—NEUROLOGIC FINDINGS

Case Number	Third Nerve					Fourth Nerve	Fifth Nerve	Sixth Nerve	Seventh Nerve		Ninth Nerve	Tenth Nerve	Kernig's Sign	Babinski Sign	Reflexes		Tremor	Parkinsonian-Like Syndrome	Muscular Twitchings	Nystagmus	Speech and Writing Defect
	Ptosis		Pupillary Changes		Extraocular Palsies				Right	Left					Deep	Superficial					
	Right	Left	Right	Left																	
1	+	+	0	0	0	0	0	0	0	+	+?	0	0	0	0	0	+	0	0	0	+
2	0	+	0	+	+	+	0	0	0	+	0	0	0	0	+	0	0	0	0	0	0
3	+	?	0	0	+	0	+?	0	0	+	+	+	0	0	+	0	+	+	0	0	+
4	0	0	0	0	0	+?	+	0	0	0	0	0	0	0	0	0	+?	0	+	+	0
5	0	+	0	0	+	0	0?	0	0	+	0	0	0	0	0	0	0	0	+	0	0
6	+	+	0	0	+?	0	?	0	+	0	+?	0	0	+?	+	0	+	+	0	0	+
7	0	0	0	0	0	0	0	+	0	0	0?	0	0	0	0?	0	0	0	0	0	+
8	+	+	+	+	+	0	0	0	+	0	0	0	0	0	+	0	+	+	0	+	+
9	+	+	+	+	+	0	0	0	0	0	0	0	0	0	0	0	+	+	0	+	+
10	0	0	0	0	0	0	0	0	0	+	0	0	0	0	0	0	0	0	0	0	0
11	0	0	0	0	0	0	0	0	0	+	0	0	0	0	+	0	0	0	0	0	+
12	+	+	+	+	+?	0	0	0	+	+	0	0	0	0	0	0	+	0	+	+	+
13	0	0	0	0	0	0	0	0	+?	0	0	+	0	0	0	0	++	0	0	0	+
14	0	0	0	0	0?	0	0	+	0	0	+	0	0	0	+	0	+	0	0	0	0
15	0	0	0	0	+	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
16	+	+	0	0	+	0	0	+	+?	0	0	0	0	0	0	+?	++	0	0	0	0
17	+	+	+	0	+	+	0	0	+	0	0	+?	+	+?	+	0	++	+	0	+	0
18	0	0	+	+	0	0	+	+	0	0	0	0	0	0	0	0	+	0	+	+	0
19	0	0	+	+	0	0	+?	0	0	0	+	+	0	0	0	0	++	0	0	0	+
20	+	+	+	+	+	0	0	0	+	+	+	+	0	0	+	+	+	0	+	+	0
21	+	+	0	0	0	0	0	0	0	0	+	+	0	0	0	0	+	0	+	0	+
22	0	0	+	0	0	0	0	0	0	0	0	0	0	0	0	0	+	0	0	0	+
23	+	+	+	+	0	0	0	0	0	0	+?	0	0	0	0	0	0	0	0	0	+

present in thirteen cases. In three it was unilateral, in ten bilateral. Pupillary changes were noted in ten cases; unilateral in three. Eccentricity of pupils, reaction to light and not to accommodation and rigidity of pupils were the common form of alteration. An unusual feature of the pupillary changes was their variability and inconstancy.

The fourth nerve was involved in three cases, the sixth in four.

The fifth nerve was involved in five cases.

Seventh nerve: Thirteen cases showed facial weakness; weakness rather than paralysis was the rule. In only two cases was the seventh nerve involvement bilateral.

The ninth nerve was involved in eight cases, and the tenth in six, evidenced by difficulty in swallowing, difficulty in phonation, hiccup, etc.

The deep reflexes were altered in eight cases and the superficial in two. The usual change was inequality of the two sides. Babinski's sign was seen in only two cases, and in both the finding was equivocal.

Tremor was present in sixteen cases, usually coarse and irregular. In four cases it was marked. It involved, in various cases, practically the entire musculature of the body.

A parkinsonian-like syndrome with mask-like facies, propulsive gait, rigidity, etc., was present, in part, in five cases.

Hiccup was present in two cases. In one it persisted for over a year.

Areas of hyperesthesia were present in two cases (9 and 14).

Nystagmus, usually irregular, was seen in seven cases.

Speech and writing defects were noted in thirteen cases.

The usual abnormalities in writing were tremor and carelessness. The speech was usually thick, slurring and careless. Distortion was absent.

Peripheral neuritis, optic neuritis and masseter tenderness were each observed once.

Muscle twitchings were seen in six cases.

LABORATORY FINDINGS

The spinal fluid findings are shown in Table 4. Mention of pressure is omitted because in routine lumbar punctures we do not use a pressure gage. Also, so much depends on the position of the needle in the spinal canal.

The methods used for testing for globulin were those of Ross-Jones and Pandy. The globulin is reported as being from 1 to 6 +, 6 + indicating the heaviest cloud that is ordinarily obtainable in a clear fluid, and 1 indicates a trace. The Wassermann test was made with three antigens and double and quadruple quantities of spinal fluid were used with antigen (B) cholesterinized beef heart extract. In the colloidal gold curves we used gold solutions prepared according to the method of Miller, Fulton, Brush and Hammer.[10]

Cell Counts.—We regard over 10 cells per cubic millimeter as definitely pathologic and counts greater than 5 cells per cubic millimeter as suggestive of pathologic change. In all of our cases, except four, there have been over 10 cells. The type was usually small mononuclears. Cell counts as high as 493 per cubic millimeter were encountered, with a usual increase of from 15 to 30. Of the four cases showing less than 10 cells, the patient in Case 4 was seen in the third

TABLE 4.—SPINAL FLUID FINDINGS

Case Number	Duration of Disease	Character of Fluid	Cell Count	Wassermann Reaction*					Colloidal Gold Curve	Globulin Test	
				A	B	C	2	4		Ross-Jones	Pandy
1	8 days	Clear	110	0	0	0	0	0	1122100000	++	+++
	17 days	Clear	90	0	0	0	0	0	1112200000	++	++
	52 days	Clear	18	0	0	0	0	0	1222210000	++	++
2	4 months	Clear	10	0	0	0	0	0	1222100000	+	+
3	17 days	Clear	19	0	0	0	0	0	1233200000	(+)	(+)
4	2 or 3 months	Bloody	...	0	0	0	0	0			
	2½ or 3½ mo.	Clear	5	0	0	0	0	0	2322100000	+	+
5	55 days	Clear	38	0	0	0	0	0	4432210000	+++	+++
6	42 days	Clear	5	0	0	0	0	0	0222111000	++	++
7	5 months	Clear	21	0	0	0	0	0	1222100000	+	++
	5½ months	Clear	6	0	0	0	0	0	1233310000	++	++
8	6 months	Clear	0	0	0	0	0	2	5555555420	++++	++++++
	6 months	Clear	32	0	0	0	0	0	5555555421	++++++	++++++
	7 months	Clear	472	0	0	0	0	0	1444454210	+++++	+++++
	7 months	Clear	493	0	0	0	0	0	5555555220	+++++	++++++
	7 months	Clear	26	0	0	0	0	0	4444453210	++++++	++++++
	7½ months	Clear	43	...	...	...	...	...	0211244000	++++	++++
	10½ months	Clear	27	0	0	0	0	0	11123331100	+	++
9	46 days	Clear	14	0	0	0	0	0	1100000000	(+)	++
10	13 days	Clear	207	0	0	0	0	0	1111000000	+	+
	20 days	Clear	22	0	0	0	0	0	1111000000	+	+
	27 days	Clear	17	0	0	0	0	0	1112100000	+	+
11	9 months	Clear	34	0	0	0	0	0	1122210000	++	+++
12	15 days	Clear	31	0	0	0	0	0	1122211000	+	+
13	1½ years (?)	Clear	22	0	0	0	0	0	....	+	+
14	18 days	Clear	30	0	0	0	0	0	1222100000	++	+++
	52 days	Clear	17	0	0	0	0	0	5522110000	++	+++
	89 days	Clear	12	0	0	0	0	0	3332110000	++	+++
15	4 months	Clear	3	0	0	0	0	0	1122100000	+	0
16	30 days	Clear	37	0	0	0	0	0	1110010000	+	++
17	39 days	Clear	?	0	0	0	0	0	1111100000	0	0
	43 days	Clear	?	0	0	0	0	0	1111100000	0	0
	50 days	Clear	48	0	0	0	0	0	1222200000	++	++
	60 days	Clear	?	0	0	0	0	0	1111000000	0	(+)
	70 days	Clear	?	0	0	0	0	0	1111222100	+++	++++
	113 days	Clear	15	0	0	0	0	0	2222100000	+	++
18	64 days	Clear	53	0	0	0	0	0	1211000000	++	++
19	60 days	Clear	21	0	0	0	0	0	1112210000	(+)	++
20	22 days	Clear	19	0	0	0	0	0	2322100000	++	++
	25 days	Clear	40	0	0	0	0	0	3443532111	++	++
	48 days	Clear	3	0	0	0	0	0	2231100000	+	+
	63 days	Clear	1	0	0	0	0	0	0111000000	+	+
21	6 days	Clear	16	0	0	0	0	0	0001110000	+	(+)
22	120 days	Clear	0	0	0	0	0	0	4433211100	++	++
23	150 days	Clear	6	0	0	0	0	0	2445221100	+	+

* A, B, C, refer to antigens; 2 signifies double and 4 quadruple quantities of spinal fluid used with antigen B.

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[10] Miller, Fulton, Brush and Hammer: A Further Study of the Diagnostic Value of the Colloidal Gold Reaction, Together with a Method for the Preparation of the Reagent, Bull. Johns Hopkins Hosp. **26**: No. 298 (Dec.) 1915.

month of the disease; the patient in Case 22, who was seen in the fifth month, showed globulin increase and a modified paretic curve.

The study of the cell counts has shown such a uniform increase that we are loath to make the diagnosis of encephalitis without this finding. On the other hand, the cases reported from the early English epidemic did not show the cell increase, and it has been found from study of repeatedly taken specimens that the counts vary, and when the disease goes on to recovery the count is likely to fall, although not necessarily paralleling the course of improvement (Cases 1, 10, 14, 17 and 20). This fall in the cell count may explain the absence of cell increase in four cases.

Globulin Increase.—In this clinic any increase greater than a trace is regarded as suggestively pathologic. In every case but one, more than a trace of globulin was found.

Colloidal Gold Curve.—No distinctive type of colloidal gold curve has been discovered. Many types of curve have been seen—paretic, modified paretic and high and low syphilitic zone curves. So far no menigitic curves have appeared. The type of curve was not constant for any one case on repeated examination. Although the types of gold curve are not specific, we feel that a positive curve (no curve showing less than a 3 has been regarded as abnormal) offers valuable confirmatory evidence when present. Nine cases showed definite pathologic alterations.

Wassermann Reaction.—In only one case was there any fixation, namely, a slight fixation in quadruple quantities on one occasion in Case 8.

White Blood Cell Counts and Differential Counts.—Slight leukocytosis has been the rule in our cases (from 10,000 to 17,000). This increase has persisted in some cases for several months. The differential counts have shown no pathologic alterations.

CASE 1.—*History.*—M. K., aged 40, white, married, a housewife, was admitted to the hospital, Feb. 20, 1920, and discharged, April 9, 1920. She "always feels drowsy; dizzy at times; blurred vision; nervous." The family history was negative. Menopause began in December, 1919. Since that time she had been somewhat worried. She had been a cheerful, easy-going person, with an interest in people and a real fondness for them.

The onset of the present illness was sudden on Feb. 13, 1920. She awoke feeling dizzy and faint. Later, the same day, she complained of seeing double. She staggered when trying to walk and had difficulty in going up and down stairs.

During the week before admission, she had occasional headache and complained of general weakness. Vision became more and more blurred so that she was unable to read or write. At times she wept, at other times she laughed and sang on the slightest provocation. Once she became hilarious, said she felt fine, that everything would turn out all right and that nobody need worry about her. Three days after onset the eyelids showed a tendency to droop, and this became more marked with the progression of the disease. Until the onset of ptosis she was sleepless. After that she was drowsy and frequently fell asleep while people were talking to her. Her drowsiness deepened into a stupor, but she could be aroused. She lost track of time; she imagined that she was in a hospital, and that her husband was at work (while he was at home); she thought that she saw animals, snakes, alligators, chickens and pigeons crawling on the wall. There was no fear connected with her hallucinosis. When aroused, she talked in a rambling fashion, drifting from topic to topic, and it was only with difficulty that her attention could be held. Throughout, however, when aroused, she was good-humored and said nothing was wrong with her.

The patient was very sensitive to noises and would awaken at times with a startled expression. Five days after onset she complained of difficulty and pain in swallowing, and saliva began to collect in her mouth. Her weakness continued and appeared more marked on the right side. Six days after onset herpes labialis appeared. Left facial paresis was noticed. At times she showed convulsive jerkings of the arms and legs. Her physician insisted that she had had no fever since onset.

Physical Status.—The temperature was 98.6 F., pulse rate 84 and respiration 20. The pupils were contracted and reacted to neither light nor accommodation.

The patient cooperated poorly, but apparently could not read. The eyegrounds were normal. There was marked bilateral ptosis; more marked on the right. There was no diplopia, nystagmus or strabismus. The nasolabial fold was obliterated on the left and there was weakness in closing the eyes and showing the teeth, but the forehead showed no weakness. There was impairment of sensation over the left half of the tongue which showed coarse tremor. The patient swallowed with difficulty and complained of peculiar feelings in the throat. No disturbance of sensibility was present. There was bilateral herpes labialis of the upper lip. The deep reflexes were active and equal. The Babinski and Oppenheim signs were negative. There was marked tremor of the hands and forearms in the extended position. There was some weakness of the right hand, and of the right arm and leg, more marked in the flexors. Finger-nose and heel-tibia tests were well performed, but the gait was wide and ataxic. The patient swayed in the Romberg position, but did not fall. She was drowsy during the entire examination. The blood pressure was: systolic 105, diastolic 60. The physical examination was essentially negative otherwise.

Blood Examination: The blood findings were: red blood cells, 4,880,000; hemoglobin, 78 per cent.; white blood cells, 10,880. Differential Count: Polymorphonuclears, 70 per cent.; polymorphonuclear basophils, none; polymorphonuclear eosinophils, 5 per cent.; lymphocytes, 22.5 per cent.; large mononuclears, 5 per cent., and transitionals, 2 per cent.

Spinal Fluid: The findings, February 21, on the eighth day of the disease, were: clear; slightly increased pressure; 100 cells; globulin, Ross-Jones test + +, Pandy, + + +; Wassermann reaction, negative; colloidal gold curve, 1122100000. Urine: In the urine were 0.5 gm. of albumin per liter, occasional hyaline casts and a few white blood cells. Otherwise the urine was normal.

Mental Status on Admission.—The patient was stuporous but restless; arms and legs jerked frequently; at times her face had a startled, puzzled expression, and she then would be more alert and responsive. When aroused she answered questions relevantly and directly, but tended to lose the thread of the conversation. She said she felt sleepy and described her symptoms; she "felt fine"—"like a new-born baby, only I'm so sleepy." She denied depression. ideas of reference, influence and persecution but admitted former hallucinosis She thought she was in another hospital, missed the date by a week and did not recognize the physician. Her memory showed slight date discrepancies but was, on the whole, quite good. Her speech showed hesitancy and slurring.

Course in Hospital.—Shortly after admission her temperature rose to 101 F. and fluctuated between 101 and normal for about a month; during the last two weeks it was normal. Her pulse was commensurate with the temperature. The respiration rate was normal.

Feb. 27, 1920: Stupor and drowsiness continued. At times she was disoriented and delirious, the delirium being occupational in character. Following lumbar puncture on February 21, speech became normal; the ptosis, left facial weakness and right hemiparesis disappeared. Diplopia recurred on February 23. On February 24 she suddenly jumped out of bed, ran through the corridor and when brought back to bed wept and said, "Why do I do these things"? Frequently she confused names of doctors and nurses. When aroused she was cheerful and euphoric.

March 5, 1920: During the second week in hospital all symptoms disappeared except the somnolence, which was distinctly less. She was correctly oriented and showed growing interest in the hospital activities. Lumbar puncture on the seventeenth day of disease showed a clear fluid, with 90 cells, and + + globulin. The white blood cells numbered 9,240. She had some insomnia and received barbital in small doses.

April 9, 1920: Drowsiness had completely disappeared by March 18. She was discharged, well. There was no asthenia.

Case 2.—*History.*—N. C., a white woman, aged 63, divorced, a farmer, was admitted to the hospital, March 6, 1919, and discharged, April 12, 1919. Her father had a psychosis at 50. The type was unknown. Menstruation was accompanied by severe pain and occasionally by attacks of unconsciousness. She remained single until 50, when she married a man twenty-five years her junior. She divorced him after seven years.

During the summer of 1918 she was unnaturally irritable and unreasonable. In November, 1918, she had an attack of headache, nausea and vomiting and was in a stupor for one and a half days. She improved after two weeks in bed, but similar, though milder, attacks occurred at two to three week intervals until the end of January, 1919. The attacks are said to have been accompanied by fever. They started with flushing, fever and rigidity of the neck and shoulder muscles. The pain passed from the head to the rectum and then down the legs. On Jan. 29, 1919, the patient had another attack with vomiting. Two days later she was taken to a hospital. While in hospital she was discontented, irritable, restless and antagonistic to the physician. After twelve days she went home in spite of her weakness, and seemed more contented, although she showed some oddities—she took large quantities of cathartics, bathed practically the entire day and wrote an extraordinary number of letters.

Feb. 15, 1919, at midnight, she awoke with a scream and complained of the usual pain and stiffness in the head and shoulders. Three days later she again cried out saying some one had struck her on the left side of the head and "knocked her features off." The left eye was closed; and the left side of the face appeared "starched."

From February 18 to 25 she was quite restless, complaining constantly of symptoms of the head and said she had "an iron cross in her rectum." On one occasion she held a drinking glass in her hand, declared it was worth a million dollars and refused to allow any one to take it. She acted as if she were a queen, calling for her retainers etc. She was very sensitive to noises and screamed if the bed was moved, or if she was touched. From February 25 to March 3 she was stuporous and was brought to the clinic March 6, 1920.

Physical Status.—The patient was well nourished. The right pupil reacted to light and accommodation and was regular; the left was dilated and fixed. There was marked ptosis of the left lid with inability to move the eyeball up or down. The left eye was turned to the left; apparently third and fourth nerve involvement. Repeated examination showed no papilledema. The right nasolabial fold was flatter than the left. Facial movements were normal on both sides. Taste, deglutition, phonation and general sensibility were normal. There was no tremor of the hands, face or tongue. The biceps and triceps reflexes on the left were more active than on the right. All other deep reflexes were active and equal. The Babinski sign was negative. There was some stiffness of the neck muscles. Speech and writing were normal. The cardiovascular system was normal except for hypertension, the systolic blood pressure being 175 and the diastolic 115. Physical examination was essentially negative except for infected hemorrhoids. A roentgenogram of the head showed "areas of increased density in frontal region; sella normal." A roentgenogram of the sinuses was negative.

The urine was negative except for a moderate number of white blood cells. The spinal fluid was clear with 10 cells; globulin +; colloidal gold curve 1222100000. The Wassermann test was negative with three antigens in the blood and spinal fluid.

Mental Status on Admission.—The patient was drowsy and complained of being weak and cold. When aroused she was alert and at first answered questions distinctly and relevantly. She described her trip to the hospital but soon began to tell about wandering in the mountains and holding meetings with the negroes. She seemed quite jovial and said she felt happy and contented; she made witty remarks about the patients and seemed to understand the hospital routine, but she found fault with everything that was done for her. She fell asleep several times during the examination. She said her sister-in-law thought she was a princess and had tried to get possession of her property. She vaguely admitted having seen imaginary things.

She was disoriented as to place, said she was in another city and not a hospital, but she knew the date approximately and recognized the people around her. Her memory for remote events was fair, but poor for the recent past. She repeated seven digits correctly, but her general information was poor. She realized she was out of her head "by the things she said."

Course in Hospital.—During the first five days in the hospital the patient was delirious, complaining about everything that was done for her and demanding constant attention; she "heard her mother and sister outside the door," insisted on taking tub baths, and that she could not void. When an attempt was made to keep her in bed she struck at the nurse. Throughout this period. however, she was cheerful and good-humored in contrast to her previous period of complaints. At night she frequently rambled on in a confused fashion about negroes, etc. She forgot her meals and insisted that she had not breakfasted when she had just been served. Her complaints about nursing care and about headaches and pain in her neck and abdomen persisted until March 21. From that time on she showed increasing interest in the ward activities and her friends. She was discharged April 12, 1919, practically recovered except for third and fourth nerve palsies.

Catamnestic Data.—The patient has been well up to the present writing, but there has been little change in the left eye; the third and fourth nerve palsies persist but have improved.

CASE 3.—*History.*—S. K., white, aged 23, married, a housewife, was admitted to the hospital, Nov. 8, 1919, and discharged Dec. 2, 1919. Her father died of brain fever (?) at the age of 28. She was friendly, sociable, and showed no signs of instability.

For two months preceding the onset of the present illness the patient had been run down and complained of indigestion. Three weeks before admission she had a bad cold and complained of severe headache and fatigue. Two weeks before admission she was frightened by a burglar and did not get out of bed again. She lay with eyes closed and did not speak. She was taken to a general hospital. There she lay with eyes partly closed, seemed indifferent to her surroundings, ate only when fed and voided only when taken to the toilet. At times she appeared apprehensive. There was some tremor of the fingers with general rigidity. Her voice was low and monotonous, and she obeyed commands in an automatic fashion. The rectal temperature was 100 F. She had 17,500 white blood cells per cubic millimeter, and a few days later 13,000. She became restless, insisted noisily on getting out of bed, and was admitted to the clinic, Nov. 8, 1919, with a diagnosis of hysteria and dementia praecox.

Physical Examination.—The patient was pale and cyanotic with a masklike expression. She was drooling and could not swallow. She lay with eyes closed. The general muscle tonus was increased, more so on the left. The head was retracted but could be flexed without pain. Kernig's sign was negative. She could raise her eyelids only part way. Ocular movements and optic disks were normal. The patient could open the mouth only slightly and could not move the jaw laterally. She could not wrinkle the forehead or show the teeth. She swallowed with difficulty. Her voice was low and monotonous, and there was distinct difficulty of articulation. The tongue could not be protruded beyond the teeth. There was no disturbance of sensibility. She complained of pain in the left arm and leg and refused fully to extend them, but no special tenderness could be made out. All deep reflexes were active, but those on the left side seemed to be more active. The superficial reflexes were active, there was no Babinski sign. The patient's gait was shuffling, with short steps. There was a coarse, bobbing tremor of the head with coarse tremor of the hands and jerking movements in the right arm.

The temperature was 100 F., pulse rate 76 and respiration 22. The remainder of the physical examination was negative. The white blood cells numbered 14,000 and included: polymorphonuclears, 82 per cent.; polymorphonuclear basophils, 1 per cent.; polymorphonuclear eosinophils 3.5 per cent.; small mononuclears, 12 per cent.; large mononuclears, none, and transitionals, 1.5 per cent. The spinal fluid was clear, with a trace of globulin and 19 cells; colloidal gold curve 1232200000. The culture was negative. The urine was negative except for acetonuria.

Mental Status on Admission.—The patient was restless, refused to stay in bed and wandered about the ward. She answered questions simply and to the point, and asked frequently, "Won't you give me some medicine to put me to sleep"? She described her mood as "all right." She was correctly oriented, and her memory was good for recent and remote events. Retention, calculation and general information were adequate. She knew she was sick.

Course in Hospital.—The patient's restlessness persisted, and she could not be kept in bed. On November 9, she shouted and screamed and attempted suicide by tying a shoestring around her neck. Her temperature rose to 101 F., and her pulse rate was 140. She had to be fed through a tube because of dysphagia. Her general rigidity and tremor increased. On November 10, she could swallow food, restlessness decreased, and she was more cooperative. Right ptosis was marked, the tongue deviated to the right, and the left side of the mouth was less active than the right. On November 12, she complained of drowsiness and double vision, but the rigidity had disappeared. The temperature fell to normal on November 14, and remained so. Her behavior showed no abnormality except for tearfulness when she was not allowed to mingle with other patients. By November 22 behavior had become normal, and all neurologic signs had disappeared. She was discharged as well on December 2.

The diagnosis of hysteria had been made outside because of the great improvement of the ptosis and swallowing when the patient was emphatically urged. The diagnosis of dementia praecox was undoubtedly due to the haste with which any semblance of stupor is taken for catatonia.

CASE 4.—*History.*—F. W., white, aged 44, married, housewife, was admitted to the hospital, March 24, 1919, and discharged, Sept. 4, 1919. Her mother was a "chronic invalid" and had migraine. A maternal uncle had "mental trouble," and one brother committed suicide at the age of 19. The patient was a frail, irritable child. She began to imitate the invalidism of her mother and brother at an early age and became whimsical about her food. Menstruation was painful and accompanied by migraine. The patient had been hypochondriacal with innumerable complaints for years, had consulted many physicians and had had three pelvic operations without relief.

During the summer of 1918 there was an aggravation in symptoms, and the patient made some suicidal remarks. In January, 1919, she had an acute febrile illness for three days, in which her temperature was 102 F. Following this she was confined to bed and complained constantly of pain in the head.

In February she had an attack of tonsillitis (?), after which she felt she would fall forward; her jaw would drop, her hands and arms quivered when she raised them. On March 14, 1919, a jerking movement of the eyeballs was noticed, which increased on attempt at fixation. She began to fear blindness and insanity, and her hypochondriasis increased with constant demands for reassurance and attention. Since onset, she complained of headaches and vertigo.

Physical Status on Admission.—The patient looked sick and showed marked undernutrition. The temperature was normal. The glands at the angle of the jaw and the posterior cervicals were definitely enlarged.

The pupils were regular and reacted to light and accommodation. There was slight strabismus of the left eye; but the most striking feature was a most extraordinary lightning-like jerking of the eyeballs with rapid excursions in all directions, occurring spontaneously and lasting from one to two seconds. The otologist reported "rapid nystagmus, irregular in action and time." Lateral deviation checked it somewhat. Irrigation of the right or left ear with cold water caused marked vertigo and nausea after about one half the normal time. Oscillation increased after irrigation. Impression: marked over-irritability of the vestibular apparatus on both sides. Otherwise the physical examination was negative, except for some jerking movements of the muscles of the neck and a staggering gait. The Romberg sign was negative. Finger-nose test was well performed. The blood pressure was: systolic, 120; diastolic, 80. The eyegrounds showed no sclerosis. The white blood cells numbered 6,000 and the red blood cells 4,500,000; hemoglobin, 80 per cent. The urine was negative. Lumbar puncture, April 1, 1919, brought bloody fluid. Another on April 15 showed 5 cells. The globulin was normal; the Wassermann test was negative; colloidal gold curve 2322100000.

Mental Status on Admission.—The patient was correctly oriented, and her mental status showed no abnormality except persistent hypochondriasis—fear of insanity, dizziness, fear of blindness, pain all over the body, gastro-intestinal complaints, etc. Her judgment and insight were good.

Course in Hospital.—The patient began to improve shortly after admission. The nystagmoid movements, also observed during sleep, gradually decreased. On discharge the patient was practically well. The eye movements were observed only in an extreme lateral position. Vertigo was complained of only at rare intervals, and the hypochondriasis had disappeared almost completely, although at times the patient wanted reassurance.

The fundamental difference from any known type of nystagmus at first gave rise to the question of hysteria. But for hysteria it appeared too far beyond the possibility of imitation, and the accompanying twitches and rigidity of the neck and the entire attitude of the patient spoke against a purely psychogenic origin. Momentary eye tantrums without any conscious component except dizziness would describe the type better than the term nystagmus.

Case 5.—*History.*—E. G. S., white, aged 27, married, a farmer, was admitted to the hospital, April 22, 1919, and discharged, June 11, 1919. His maternal grandmother was insane. His personal history was negative.

March 1, 1919, when he arose, the patient complained that everything was black before his eyes. On March 2, he felt "dazed" and dizzy; he saw double and staggered when he walked. He went to bed and for ten days was dull and talked incoherently about his farming. He could be aroused only with difficulty and complained of headache. During the third week of his illness he became restless, agitated, talkative and complained of unbearable headaches. He thought there were wheels in his head, and that his head was divided into several parts.

At the onset of the restless period his mouth became drawn to the right, and his eyes "rolled up and out"; there were twitchings on the left side of his face, and his whole body jerked. For the five weeks before admission there was difficulty in chewing, and weakness of the right arm. The patient became over-talkative, and was inclined to be obscene and to curse. The staggering gait continued.

Physical Status on Admission.—The patient was well nourished. There was ptosis of the left eyelid, and the pupils responded sluggishly to light but briskly to accommodation. There was no strabismus or nystagmus. The fundi were normal.

There was definite paresis of the left side of the face. No disturbance of deglutition or sensibility was present. The grip was weak and there was some weakness of the flexors of the arm on the right. Deep and superficial reflexes were equal and active. There was no Babinski sign and no tremor. The gait was reeling. The heel-knee and finger-nose tests were poorly performed.

There was swaying in the Romberg position and a tendency to fall backward. The blood pressure was: systolic 135, diastolic 90. The urine was normal. The blood Wassermann reaction was negative. The white blood cells numbered 10,300, which included polymorphonuclears 75 per cent., polymorphonuclear eosinophils 3 per cent., small mononuclears 17 per cent., large mononuclears 3 per cent., and transitionals 2 per cent. There was 78 per cent. hemoglobin. The spinal fluid was clear, with 38 cells; globulin + + +. The Wassermann reaction was negative; colloidal gold curve 4432210000. The temperature was normal on admission. A roentgenogram of the head showed normal sella, but small dense shadows just above the temporal fossa.

Mental Status on Admission.—The patient was alert and cooperative; he showed a push of talk without distortion. He said he felt well and cheerful, but he wept when thinking of his friends. His memory was good, but he refused to cooperate in retention. He was correctly oriented and showed some insight into his condition.

Course in Hospital.—The patient's temperature fluctuated between normal and 101 F., until May 28, when it fell to normal and so remained. The pulse at times was irregular and rapid (130). The talkativeness and restlessness continued until June 5, 1919. He was cheerful and in a good humor always, but from May 2 to 11 he would burst into laughter or tears without adequate stimulus. He thought at times that his head was unclean and insisted on washing his hands frequently. He also complained of a bad odor in his room. For three days in the middle of May he was drowsy and sleepy. Facial weakness, ptosis and gait began to improve about the middle of May and progressed steadily to complete recovery, except for slight left facial weakness.

Case 6.—*History.*—J. B., white, aged 46, married, a merchant, was admitted to the hospital, March 3, 1919, and discharged, March 27, 1919. His family history was negative. When 27 years of age, the patient had business reverses and was depressed and tearful for a short period but soon recovered. He was a hard worker, even-tempered, uncomplaining, but seclusive. His interests had been limited.

Six weeks before admission he had had a severe cold. Five weeks before admission he began to lose sleep and to be nervous. Three weeks ago he became drowsy and complained of pain in the back of the head and neck. Once he said he was sad, and that he would die. His drowsiness deepened into stupor, and for the week before admission he could not open his mouth or eyes or talk plainly and complained of "drawing" in his eyes. He also had some ringing in one ear.

Physical Examination on Admission.—The face was masklike, and there was marked rigidity of the entire body. There was ptosis of both lids, more marked on the right. The pupils reacted promptly to light and accommodation. Ophthalmoscopic examination was negative. There was some dysphagia and articulation of the labials was difficult. All deep reflexes were overactive and the superficial ones were easily elicited. There was slight tremor of the extended hands. The gait was spastic. The patient complained of pains in the jaws. The blood pressure was: systolic 142, diastolic 90. The white blood cells numbered 8,600; hemoglobin, 89 per cent. The blood Wassermann reaction was negative. The spinal fluid was clear, with increased pressure, 5 cells; globulin, + +; colloidal gold curve, 0222111000. The urine was negative. A roentgenogram of the head showed that the posterior clinoid was irregular and an area of increased density in the cerebellar region.

Mental Status on Admission.—The patient's actual alertness was in striking contrast to his appearance of stupor. He answered questions directly and relevantly and was interested in the ward activities. He said his spirits were good, although at times he was sad about being away from home. He was correctly oriented. His memory was good for recent events, and fairly good for remote events. He corrected his mistakes in dates. His retention was satisfactory. Calculation was excellent. He realized that he was sick mentally.

Course in Hospital.—The patient's temperature ranged between normal and 100 F. until the seventh day in the hospital, when it fell to normal and so remained. On the third day the face was drawn to the left—apparently a right-sided weakness. He complained of headache from time to time; the rigidity persisted. He maintained that he could not put out the tongue. On March 14, there was a positive Babinski sign on the right, and the reflexes were more active on the right. On March 16, the tongue deviated to the left, and there was impairment of taste over the right anterior part of the tongue. The ocular movements were quite sluggish. On March 27, 1919, the facial weakness had disappeared; the rigidity had also disappeared, although the reflexes were brisk. His mind was quite clear, and his memory showed no defects.

CASE 7. *History.*—J. T., white, aged 42, widower, a merchant, was admitted to the hospital, Nov. 29, 1919, and discharged, Dec. 26, 1919. His family history was negative. He was normal at birth and his development was normal. He was a successful boat captain. He was married, March, 1918, and his wife died in November of the same year. The patient apparently led a normal, happy life.

In November, 1918, the patient had influenza. July 1, 1919, he began to feel tired, weak and "achy," and had frequent periods of drowsiness, often falling asleep while at work or driving his automobile. Speech was thick, and he spoke in a low tone, at times in a whisper. At times he could not swallow. His symptoms increased in severity, and at times he was delirious; he imagined he was talking to his dead wife and sister, had feelings of unreality—he thought his legs were those of some one else, etc. He lost 60 pounds in weight. In November, 1919, he complained of pain across the back and legs. He is said to have had fever.

Physical Examination on Admission.—His temperature was 100 F., his pulse rate 90 and respiration 20. He showed obvious loss of weight. There was some tenderness in the sacro-iliac region and tenderness in the thigh and calf muscles. The pupils reacted to light and accommodation. There was no nystagmus or diplopia. Ophthalmoscopic examination was negative. The patient limped as he walked and used a wide base. He tried to shield the left leg.

His writing was tremulous and uncertain, and his speech was distinct but low in tone and monotonous. There was some thickening of the peripheral vessels. The blood pressure was: systolic 130, diastolic 60.

The urine showed a large quantity of albumin; otherwise it was normal; phenolsulphonephthalein test 50 per cent.; urea nitrogen of blood 25.5 mg. per 100 cubic centimeters of blood.

There were a left inguinal hernia and hydrocele. The red blood cells numbered 12,120, including: polymorphonuclears 71 per cent., polymorphonuclear eosinophils 3 per cent., lymphocytes 19 per cent., large mononuclears 5 per cent., transitionals 2 per cent.; hemoglobin 75 per cent. The spinal fluid was clear, under increased pressure, 21 cells, globulin ++. The Wassermann reaction was negative; colloidal gold curve 1222100000. The roentgenogram showed slight infectious arthritis of the spine.

Mental Status on Admission.—Except for changing position frequently on account of pain, the patient showed no abnormality in behavior. His talk was relevant and direct. He said he felt "blue and downhearted" over his condition. He admitted the hallucinatory experiences noted in the foregoing, but insisted that it was dream material. He was correctly oriented, and his memory for recent and remote events was good. Insight into his illness was good.

Course in Hospital.—Five days after admission the patient became delirious, thought he was in a boat, that his food was poisoned, that men in his room were trying to kill him, etc. He imagined that he was in a heavy fog and could not get through it; he was restless, refused to stay in bed and voided on the floor. Delirium alternated with stupor until the time of his death.

His temperature fluctuated between 99 and 101 F. until three days before death, when it rose to 104 F. On December 23, there were signs of bronchopneumonia. The right eye deviated to the right. The urine showed heavy albumin and granular casts. The leukocytes numbered 18,920 on the 24th, the temperature was 104 F., respiration 44, and pulse 124. Blood cultures and spinal fluid cultures were persistently negative. On December 26, the patient died. Necropsy examination was refused.

Case 8.—*History.*—F. J. M., aged 53, a mail carrier, married, was admitted to the hospital, Sept. 25, 1919, and died, March 4, 1920. His family history was negative.

In February, 1919, the patient had influenza (?). He was in bed only one week, but afterward he felt weak and nervous, in spite of which he insisted on remaining at work until April, 1919, when he had a ten day attack of nausea, vomiting and headache. He became delirious and remained so until June, 1919, at which time he seemed well enough to return to work. After a few days he had to give up because he made so many mistakes in his work. Gradually he became more and more confused and increasingly drowsy and delirious.

Physical Examination on Admission.—The patient showed loss of weight and a temperature of 102 F. There was a gross, coarse tremor of the head and extremities. The tremor decreased with purposive movements. The left pupil was larger and oval. Both reacted slightly to light but well to accommodation. There was nystagmus on lateral deviation, right ptosis and weakness of the right side of the mouth. The tongue showed coarse tremor but no deviation. Sometimes backward, and at other times forward propulsion was noted. The deep and superficial reflexes were equal and active. There was swaying and falling in the Romberg position. Speech was slurring, careless and tremulous. Writing was tremulous and careless. The blood pressure was: systolic 158, diastolic 90. The urine was normal. The white blood cells numbered 8,800; hemoglobin, 95 per cent. The spinal fluid was clear, under increased pressure, contained no cells, globulin + + + +. The Wassermann reaction was negative except for slight fixation in quadruple quantities; colloidal gold curve 5555555420. Spinal fluid culture was negative. A roentgenogram of the head showed the intracranial vessels to be markedly engorged and tortuous. There was an area of increased density above, and posterior to, the temporal fossa. The pineal gland was calcified.

Mental Status on Admission.—The patient lay quietly in bed and appeared to be in a good mood. He made occasional attempts to get out of bed. He could not identify the direction of passive movements. When asked to carry out commands he merely repeated them, but did nothing. He said he felt mixed up. He responded slowly to questions and seemed to have difficulty in understanding them. He described his spirits as good, and said that he was happy, but he wept as he said it. He could give no reason for weeping. He was completely disoriented and when given an opportunity confabulated freely. His memory was poor for remote and recent events, and he showed perseveration in dates. His immediate retention was good (seven digits), but he forgot test phrases in a few minutes. His calculation and general information were poor.

Course in Hospital.—The patient's physical condition varied from time to time until death. The temperature ranged from subnormal to 103 F., usually from 99 to 101 F. Frequent blood and spinal fluid cultures were negative. He had been incontinent since admission. The neurologic status varied. The size of the pupils changed, and frequently the right would be larger than the left. Ptosis became more marked on the left. Occasionally ankle and patellar clonus could be obtained on the right, and the right deep reflexes were increased. The spinal fluid findings varied but always showed increase in cells and a paretic type of gold curve. Examination for tubercle bacilli gave a negative result. The Wassermann test remained permanently negative. His inanition increased, and large bed sores developed. He died March 4, 1920, from respiratory failure. Necropsy examination was refused.

The mental condition varied, but, except for occasional periods of brightness, showed no essential change. He constantly maintained that he felt fine, and that there was nothing wrong. He continued to confabulate freely. February 14, he stopped speaking and became stuporous.

Case 9.—*History.*—T. W. S., white, aged 28, married, a crane engineer, was admitted to the hospital, March 2, 1920, and discharged, April 5, 1920. His family and personal history were negative.

Onset occurred Feb. 16, 1920, with pains in the back and face, especially over the forehead and jaw. The temperature was between 101 and 102 F. Four days later the antrum and frontal sinus on either side punctured and yielded pus (?). On February 23, tremor involving first the arms and then the head and body appeared. At the same time the patient became delirious. Three days later he complained of seeing double; there was paresis of the left internal rectus. The temperature was slightly elevated until admission, when it was 100 F.

Physical Status on Admission.—The temperature was 100.5 F., pulse rate 100 and respiration 20. The patient was well nourished, somnolent and weak. The pupils reacted promptly to accommodation but sluggishly to light. There were vertical and horizontal nystagmus and weakness of both eyelids. The eyegrounds were normal.

There was increased tonus in the sternocleidomastoids, and the trapezii seemed weak. The superficial and deep reflexes were normal. There was some hyperesthesia in the lower part of both arms. There was no Kernig's sign. There was coarse tremor on intention and occasional twitchings in the back and neck. The blood pressure was: systolic 135, diastolic 90. The urine was normal. The white blood cells numbered 16,360; hemoglobin 84 per cent. The blood Wassermann test was negative. Lumbar puncture yielded bloody fluid (fresh blood). Examination of the sinus showed no abnormality.

Mental Status.—The patient lay quietly in bed. He was somnolent but could be easily aroused, and would then answer questions. He tended to drift into talk about his occupation, at times growing excited about it, and he gave the impression of attention difficulty. He described his mood as good. He was oriented for place, but not for time or person. His memory for remote and recent events was not accurate but showed no more distortion than the delirium would produce. Retention, calculation, general information and judgment were satisfactory when attention could be fixed. Speech and writing showed carelessness and slurring.

Course in Hospital.—On March 12, the patient's condition was practically the same as on admission, except that he was brighter. He was overproductive in his talk when started, but showed no especial distortion. He complained of pain in the back and shoulders. The patient began to improve rapidly, and by March 30 was almost well. The following abnormalities were, however, noted: asthenia, pain, soreness and weakness of the right arm, right pupil larger than the left, slight tremor of the arms and legs. The spinal fluid at this time was clear; it contained 14 cells; globulin +. The Wassermann reaction was negative; the colloidal gold curve 1100000000. The temperature remained normal after the eighteenth day in the hospital.

CASE 10.—*History.*—L. B., white, aged 26, a widower, a huckster, was admitted to the hospital, March 20, 1920, and discharged, April 23, 1920. One uncle was insane. The patient has always exaggerated any physical complaint and was regarded by his family as nervous and unstable. October, 1918, the patient fractured the base of his skull. November, 1918, there was decompression, after signs of increased intracranial pressure had developed. Recovery was uneventful.

In March, 1920, the patient began to have a sense of pressure in the head with some bulging in the decompression wound. On March 14, he had a "dizzy spell" with vomiting. From March 4 to 17 he was unable to sleep, walked the floor, humming and singing and said he felt well but realized that he was sick. On March 18 he felt weak, restless and nervous, and there was some jerking of the limbs, head and eyes. Admitted to the surgical service with a temperature of 102 F., he was so restless and talkative that he was transferred the following day to the psychiatric service.

Physical Status on Admission.—The temperature was 101.6 F. Except for some slight weakness of the right lip and the old decompression scar, the physical examination was negative. The white blood cells numbered 16,900 on the day of admission and 10,600 on the following day. The patient had taken practically no fluid for four days before admission. Examination of the urine was negative. The spinal fluid was clear, containing 207 cells; globulin +. The Wassermann reaction was negative; colloidal gold curve 1111000000. The spinal fluid culture was negative.

Mental Status on Admission.—The patient was alert but somewhat restless. When allowed to talk, he showed overproductivity but no distortion. He described his mood as good, was correctly oriented and memory, retention, general information, judgment and insight were excellent.

Course in Hospital.—The patient slept well after the first night and showed rapid improvement in his mental symptoms. His talk became normal after a few days, and except for oversensitiveness to noise, and asthenia, he showed no abnormality. He left the hospital feeling well, although there was some asthenia. Lumbar puncture showed sudden decrease in cellular elements.

Catamnestic Data: Sept. 15, 1920, the patient was unable to sleep at night but was drowsy in the daytime.

CASE 11.—*History.*—E. W., white, aged 47, married, an engineer, was admitted to the hospital, Feb. 14, 1920, and discharged, Feb. 28, 1920. His family history was negative. His personal history was also negative. He used alcohol moderately.

On April 16, 1919, the patient returned from a business trip saying he was "all in" and that he had "fallen to pieces." He drove his motor car so recklessly that his family consulted a physician. He was put to bed for six weeks. During this time he is said to have had fever as high as 104 F. At the same time left-sided weakness, involving the face and extremities, was noted. He grew quite irritable and after a forced cold sponge bath was unable to speak for two weeks, after which his behavior had been peculiar, and he had had numerous hypochondriacal complaints. Since June, 1919, his condition had remained practically stationary.

Physical Examination.—The patient was poorly nourished and showed evidence of loss of weight. The pupils were normal, and there was no nystagmus or ptosis. Deviation of the eyes to the extreme right was maintained with difficulty. There was some optic neuritis on the right; physiologic cupping was absent, and there was some exudate alongside the vessels. The left fundus showed two hemorrhagic areas. The left corner of the mouth drooped. There was no sensory disturbance. The deep reflexes in the left leg were exaggerated. The Babinski sign was negative, and there was no clonus. The superficial reflexes were normal. There was some weakness of the left leg. The blood pressure was: systolic 140, diastolic 80. The urine showed a heavy cloud of albumin and hyaline and granular casts. There was no peripheral arteriosclerosis. Phenolsulphonephthalein was normal. The temperature was 100.5 F. Blood examination revealed: hemoglobin, 60 per cent.; red blood cells, 3,500,000; white blood cells, 11,000, including polymorphonuclears, 68 per cent.; large lymphocytes, 3 per cent.; small mononuclears, 20 per cent.; large mononuclears, 1 per cent.; transitionals, 8 per cent. The blood Wassermann reaction was negative. The spinal fluid was clear, with increased pressure, 34 cells, globulin + +, Ross-Jones, Pandy test + + +. The Wassermann reaction was negative; colloidal gold curve 1122210000. A roentgenogram of the head showed "intracranial vessels very much distended and tortuous. No evidence of any localized lesion."

Mental Status on Admission.—The patient was quiet and cooperative, but spoke in stilted, bizarre fashion. At times he enforced his remarks with outbursts of swearing. This was quite pointless; for example, "You would laugh if they would get me rid of this eye trouble, which keeps me from reading and seeing, and this stomach. I would call for my wife immediately to come for me. What am I here for? Damn! I haven't syphilis. Take your damn fool tests. I am glad to be through with them. Give me my stomach and my eyes and Hell! Good God! Do I speak to the point? (Any odd notions?) I am hesitating because I want to be damn sure that I am telling exactly what is true . . . absolutely none. No unpleasant feelings, no anything at all." The patient was partially disoriented, did not recognize the physician or know the date. His memory for recent events was poor, but fair for remote. His retention was only 5 digits. Calculation and general information were poor. His writing was careless and his speech showed some slurring. He had little insight. His mood he described as good.

Course in Hospital.—The patient's temperature varied between normal and 101 F. during his stay in the hospital. Neither neurologic nor mental status showed change. He was irritable at times and frequently confused and disoriented. He lost his way about the ward and his talk remained prolix and full of circumlocution. He misused words constantly and lost the point of his talk, although it did not seem to be delirious in its meanderings. Provocative arsphenamin injections gave negative blood results. Two months after leaving the hospital the patient died. No details are known.

CASE 12.—*History.*—J. F. Y., white, aged 24, single, a clerk, was admitted to the hospital, March 5, 1920, and died, March 18, 1920. His family and personal history were negative.

The onset, which was acute, occurred Feb. 16, 1920, with neuralgia (?) of the ear. February 19 he quit work, became very weak and restless and was seized with severe pains all over the body (?) accompanied by generalized twitchings and tremor. He was treated for a "nervous breakdown."

Physical Examination on Admission.—The patient was well nourished. His temperature was 99 F., and pulse rate 104. He complained of "pains all through the body, twitchings and earache." The patient could not cooperate well, and apparently there was bilateral internal rectus weakness. There was marked horizontal nystagmus, with slow movements to the left, and bilateral ptosis. The fundi were normal. There was facial weakness on both sides, more marked on the right. There was no sensibility disturbance. Deep and superficial reflexes were equal and active. There was no clonus or Babinski sign. The abdominal muscles were held rigidly, and the abdominal reflexes could not be elicited. The Kernig sign was negative. There were occasional muscular twitchings in all of the extremities. Speech was slurring. There was coarse tremor of the extremities at times. The heart showed marked sinus arrhythmia. The blood pressure was: systolic 130, diastolic 90. There was marked pharyngitis. The urine contained numerous white blood cells and on culture gave a nonhemolytic streptococcus. Blood examination revealed 14,000 white blood cells; 80 per cent. hemoglobin; a negative Wassermann reaction; a negative blood culture; spinal fluid clear, with some increase in pressure; 31 cells; globulin +; the Wassermann reaction negative and a colloidal gold curve 1122211000.

Mental Status on Admission.—The patient was restless and tossed about as if in great discomfort. He appeared to keep contact only by effort; he answered questions readily but tended to drift off deliriously. He was disoriented for time and only approximately oriented for person. He described his mood as "all right." His memory was impaired but no more than the delirium would account for. Speech and writing showed slurring and difficulty in attention.

Course in Hospital.—The patient's temperature rose to 102 F. and remained elevated until his death, when it was 105. On the ninth day of the disease the pupils became rigid and a corneal ulcer appeared on the left. Severe cystitis developed and probably pyelitis. He became delirious and could not be kept in bed. At times he complained of seeing double.

On March 16, the patient began to fibrillate, and he died on March 18. The general tremor and abdominal rigidity persisted until the end. Necropsy examination was refused.

CASE 13.—*History.*—I. W. P., white, aged 40, married, was admitted to the hospital, Oct. 18, 1919, and discharged Dec. 2, 1919. His family and personal history were negative.

In the fall of 1918 the patient began to lose interest in his work and seemed tired and worn out. In September, 1918, he had severe temporal headaches accompanied by hiccup which recurred from time to time during the year.

He also complained of loss of hearing with tinnitus and had frequent dizzy spells. On two occasions during 1919 he fainted and was unconscious for half an hour. For the past year memory has been failing, but it has showed marked variations. At times he can remember well and at other times his mind seems blank. Since the middle of September, 1919, he had been very unsteady on his feet, and since the last week in September he had been incontinent.

Physical Examination on Admission.—The patient was sleepy and fell asleep during examination. The eyes showed no abnormality with the exception of some haziness of the disks. The tongue was protruded slightly to the right. There was marked tremor of the entire body, including the lips and face, which at times became violent. He was incontinent (?) whenever somnolent. His blood pressure was: systolic 120, diastolic 80. Spinal fluid findings were: increased pressure, 22 cells, globulin +, and negative Wassermann reaction. A roentgenogram of the head revealed no abnormality. The white blood cells numbered 8,200. (The differential count was normal.) There was slight prostatitis, moderate pyorrhea. Urine examination was negative.

Mental Status on Admission.—This varied markedly. When the patient was first seen he insisted on his wife leaving the room and said, "I feel as if I had spent a week in hell." He said he did not know how he felt, and then laughed. At times he was disoriented and failed to remember his examinations; he persistently wandered into the wrong rooms, and urinated in the tooth mug. He thought he heard the president of his company on the floor below. Then he lay quietly in bed and from time to time fell asleep. When aroused he smiled naturally but in a rather whimsical fashion. He answered questions for a time and then closed his eyes and refused to speak. He said his spirits were good, and his attitude suggested this. He admitted hallucinatory experiences but refused to discuss them. He thought there were detectives watching him, but there was no apprehension or distress about it. Orientation was only partial. He knew he was in a hospital but did not know the month or year. Retention was fair; he retained six digits. Calculation was poor. He joked about the examination to cover up his deficit. He showed no insight.

Course in Hospital.—His physical condition did not change during his stay in the hospital. From time to time he had periods of violent tremor which gave the impression of a chill. Hiccuping occurred from time to time and persisted for from 24 to 72 hours. His temperature was normal throughout his entire stay in the hospital. His mental condition varied markedly. He was never completely oriented, and his memory was persistently poor. Retention, calculation and insight showed no improvement. At times he was in fair contact with the environment—would talk about casual topics. He always joked when examined and tried to cover up his mistakes by bantering with the physician. At times he would pack his suit case and prepare to leave, but would not say when he was going. He was finally transferred to a state hospital near his home.

CASE 14.—*History.*—W. H. E., white, aged 24, single, a medical student, was admitted to the hospital, Feb. 2, 1920, and discharged, June 9, 1920. His father died at the age of 56 of an apoplectic stroke. His personal history was uneventful. He was shy, was diffident and moderately alcoholic.

During the fall of 1919 the patient was in better humor than he had been for years and friends remarked on his talkativeness. During January, 1920, the patient felt "off color," and his appetite was poor. Jan. 26, 1920, he had a severe headache and felt weak and tired. He gave up work on the 28th when he discovered his temperature was 102 F. He was admitted to the medical service on January 30, complaining of headache and general malaise. On February 1, he developed sharp pains just above the left great trochanter; they ran down the thigh like an electric shock. During the night he became restless and tense. He told the nurse that "these men" were trying to shoot him and spoke of electrical connections which he had to fix. He was careless about exposing himself. On occasions he wept over imagined injuries and spoke constantly about his school work. He was oriented for place, time and person, but his talk was drifting.

Physical Status on Admission.—The patient complained of shooting pains down the outer side of the left thigh, which at times ran through the buttocks. He moved restlessly in bed and cried out as if in great pain. The pupils were normal. The patient complained of diplopia. Nystagmus and strabismus were not present. The eyegrounds were normal. His tongue protruded slightly to the left. There was an area of hyperesthesia over the outer side of the left thigh from the great trochanter to the knee and particular sensibility over the ischial tuberosity. The deep reflexes were sluggish and the left knee jerk less active than the right. Ankle reflexes were absent. There was no tremor or rigidity. The urine was clear. A blood culture was negative. The white blood cells numbered 12,800, including: polymorphonuclears, 72 per cent.; large mononuclears, 2 per cent.; transitionals, 4 per cent.; large lymphocytes, 2 per cent.; small lymphocytes, 2 per cent.; small mononuclears, 20 per cent. The pulse rate was between 110 and 120; temperature, 101 F.

Mental Status on Admission.—The patient talked constantly, describing his illness in great detail. Whereas normally he talked very slowly, his talk now was extremely rapid and continuous. He was well oriented, but his talk showed delirious drifting. It was only by reminding him of his subject that he could be persuaded not to describe his delirium as facts. He tended to describe imaginary injuries, and there was much fear accompanying the episodes. He recalled with a deal of feeling a rather frank medical discussion of his mental condition which he had heard at the bedside. He insisted that he felt fine were it not for the pain. His memory was quite good when attention could be held.

Course in Hospital.—The temperature remained elevated around 100 and 101 F. for two months. There was persistent tachycardia, but the electrocardiograms showed no abnormality.

The diagnosis of "flu" made at first was changed when the clear spinal fluid under increased pressure showed 30 cells (mononuclear), globulin + +, and a colloidal gold curve of 1222100000. On February 19 some weakness of the right external rectus was discovered, and also a tremor of the eyelids and legs. The number of white blood cells varied between 10,000 and 14,000. The diplopia was absent after the first month. The spinal fluid findings on March 19 were: a clear fluid, 17 cells, globulin + + and colloidal gold curve 3332110000. The blood and spinal fluid Wassermann reactions were at all times negative.

For a period of several weeks delirium was present, especially marked at night, and, while occupational for the most part, at times marked fear and terrifying experiences were observed. For instance, he thought that there were bandits about and he was to be killed. He refused to return to his bed on one occasion because it was occupied by a woman just delivered and everything was bloody. At times he confabulated freely. Once he said that he had been down town, and on another occasion that he had been home. He worried about his school work and finances. Frequently all difficulties were solved in his delirium. He was at the horse races and won thousands of dollars. As the delirium began to pass, the patient became exacting and irritable. The slightest sound sent him into a rage, and he would relate endlessly all the discomfort of his stay in the hospital. At times he was extravagant in praise of the physician, and at others would tell at length how cases of his sort ought to be treated. Throughout the illness he was sleepless and obtained only two or three hours of sleep when given large doses of paraldehyd. Gradually the talkativeness and irritability decreased but on discharge, five months after admission, he was still overtalkative and would spend hours in discussing his illness with any one who would listen.

CASE 15.—*History.*—A. C. P., white, female, aged 29, married, was admitted to the hospital, April 20, 1920, and discharged Sept. 5, 1920. Her father was highstrung and nervous. A maternal aunt suffered from depression at the menopause. The patient was an only child, petted and spoiled by her parents, of a nervous temperament and worried over trifles. On several occasions she flew into a rage, but on the whole she controlled her temper. Her husband had difficulty in satisfying her sexually. She worried over possible pregnancy.

Feb. 1, 1920, the patient is said to have had the "flu" (?). At this time she complained of dizziness and could not focus. Following a short febrile period she did not gain strength and could not get enough sleep. Without warning she jumped from a third story window. Except for a broken heel, she sustained no injuries. The patient then admitted that she was despondent and worried and had tried to kill herself. The following day she complained of loss of memory and again jumped out of the third story window.

There were some signs of renal and visceral hemorrhage. A transfusion was performed, and she made a satisfactory recovery. She made several more suicidal attempts. Since the first fall the patient has complained insistently of loss of recent memory and retention of incidents, which she quizzed the nurses about. Morphia was required to produce sleep. Her behavior was capricious. She was easily irritated and would then pinch, slap, or bite whoever was near her. She remained markedly depressed and talked constantly of her trouble.

Physical Examination on Admission.—Physical examination was essentially negative except for evidence of the injuries and slight tachycardia. The blood pressure was: systolic 142, diastolic 88; the white blood cells numbered 5,600; the red blood cells 3,800,000; the hemoglobin content was 65 per cent. The spinal fluid findings were: two cells, no globulin, colloidal gold curve negative — 1122100000. The blood and spinal fluid Wassermann reactions were negative. .

Mental Status on Admission.—The patient lay quietly in bed but was irritable and complained about the precautions that were taken to guard her. She was well oriented. Her conversation was relevant, but there was also a lot of nagging about her treatment. She denied any suicidal attempts, was evasive about her illness, thought people were whispering about her, and said she felt blue and mixed up. Her memory showed no defect for remote events, and recent memory could not be adequately tested on account of her attitude.

Course in Hospital.—The patient's impulsive behavior continued. In the morning she was usually depressed and begged to be taken home. She made numerous crude attempts at suicide (eating a thermometer, falling out of bed, knocking her head against the bed, etc.). She confessed that her reason for the first attempt at suicide was that her husband threatened to divorce her when he discovered some marital infidelity. She resented watching and pinched and choked her nurses. She admitted her impulsive behavior but said she could not control herself. She was under high sex tension and pleaded with the physicians to caress her; later she denied this in part. She was convinced that she was pregnant in spite of the negative results of examinations, and not until the administration of luetin tablets and the beginning of menses was she convinced. She was quite willing (one suspects too willing) to accept punishment for bad behavior. Memory and insight were excellent. She gradually improved and became less impulsive than on admission.

Sept. 15, 1920: The patient had recovered completely by Sept. 1, 1920.

Case 16.—*History.*—M. C., white, aged 17, single, a student, was admitted to the hospital, April 28, 1920, and discharged, June 25, 1920. One cousin was insane. The patient was normal during childhood. Conscientious and hard working, she felt intensely about things but was not regarded as oversensitive.

On April 8, she was refused permission to attend a dance in a nearby university town. She was intensely disappointed over this, and when she also learned that her parents could not afford to allow her to return to school the following year, she wrote home that she was "utterly hopeless and miserable." A few days later she wrote that she was "sleepless, tired and melancholic" and "didn't want to live." From April 18 to 28 she had several emotional tantrums in which she wept and attempted suicide. She was excitable and felt that she was losing her mind; she insisted that she was a burden and wanted to die. On one occasion she thought she saw a lion in the room. Between outbursts she acted fairly naturally, but her outbursts occurred without apparent cause. On April 23 she saw double and on April 24 there was slight ptosis on the left. She was afebrile except for a temperature of 100 F. after a period of overactivity. The spinal fluid was clear, contained 22 cells and a slight amount of globulin. There was somnolence in the daytime but insomnia at night.

Physical Examination on Admission.—She complained of diplopia. Both external recti were slightly weak; slight right ptosis and some weakness of the right side of the mouth were present. There was a bilateral ankle clonus, but the Babinski sign was absent. The abdominal reflex was absent on the left. At times there was tremor of the entire body. She slept most of the day. The white blood cells numbered 12,680. The hemoglobin content was 85 per cent. The spinal fluid was clear; it contained 37 cells and increased globulin. The blood and spinal fluid Wassermann reactions were negative. The colloidal gold curve was 1110000000. Examination of the urine gave negative results.

Mental Status on Admission.—The patient was agitated and restless. She attempted to get to the window, apparently intending to throw herself out. She cried for her mother and seemed puzzled and fearful. She was correctly oriented. She said she could not think and did not understand anything. She repeated this again and again. She could not cooperate sufficiently to test memory, retention, etc.

Course in Hospital.—The suicidal attempts and agitation continued for several weeks, and then she became more cooperative. She appeared puzzled and easily frightened until two weeks before discharge. Her memory showed no defect, and gradually she resumed her normal activity. Except for a slight tremor, she was well on discharge.

Case 17.—*History.*—T. C. H., white, aged 36, married, a miner, was admitted to the hospital, Jan. 7, 1920, and discharged, April 21, 1920. His family history was negative. His personal history was also negative except for congenital hair lip and associated speech disorder. The patient was illiterate. The onset of the present illness occurred, Dec. 1, 1919, with frontal headache, which became progressively worse. December 15 severe pain developed in the back of the neck, which prevented sleep. Shortly before admission there was some visual disturbance, and his arms and hands became tremulous. He was dizzy and unsteady on the feet.

Physical Examination on Admission.—When first seen in the surgical service, January 7, he had amblyopia, anisocoria and left-sided ptosis. The same day he had two convulsions; his temperature rose to 104 F., the pulse was rapid, and he became comatose, restless and incontinent. The next day there was cyanosis and dyspnea, his temperature was 105 F., pulse 140 and respiration 36. Then developed incoordination of the bulbs, inequality of pupils, overactive reflexes, bilateral ankle clonus and the Babinski sign on the right. There was right facial weakness and marked rigidity of the whole body. Kernig's sign was positive. The fundi were normal. The blood pressure varied between: systolic 130, diastolic 104 and systolic 170, diastolic 112. Blood culture was negative. The red blood cells numbered 5,512,000, white blood cells 28,520, including: polymorphonuclears 82.6 per cent., small mononuclears 14.7 per cent., large mononuclears, 0.7 per cent., and transitionals, 2 per cent. The spinal fluid was normal; there was no film formation and no tubercle bacilli. On January 20 it showed 48 cells (mononuclear), globulin + +, colloidal gold curve 0111100000. The coma cleared after three days, and when the patient was admitted to the psychiatric service, January 30, the physical examination showed the same picture except for bilateral ptosis, right abducens weakness, exophoria of the left eye, jerking of the eyeballs, marked tremor of the legs, arms and hands, and a temperature of 100.6 F.; urine, negative.

Mental Status on Admission.—The patient's behavior was normal except for persistent complaint of constipation and headache, obviously hypochondriacal because both yielded to minute doses of bicarbonate of soda.

Course in Hospital.—The patient gradually improved and when discharged was normal, except for coarse tremor of the extended hands and the anisocoria. During the stay in the hospital the spinal fluid showed marked increase in globulin on February 10, with a colloidal gold curve of 1000222100, and on March 23, fifteen cells, + Ross-Jones test, Pandy, globulin, and a colloidal gold curve of 2222100000.

Case 18.—*History.*—S. S. B., white, aged 39, married, a factory worker, was admitted to the hospital, May 2, 1920, and discharged, June 22, 1920. His family history was negative. He was a competent workman. Auto-eroticism had been present since the age of 12 until the present time; it was a source of much conflict. He was married at the age of 28, and had three healthy children. He was always inclined to worry, especially over finances.

The onset of the present illness was sudden on March 1, 1920, with dizziness and headaches; he then had blurred and double vision. After one week the patient became very drowsy, and he had to be washed and fed. From March 15 to April 6, usually at night, he thought he saw animals of all kinds, elevators going up and down with people getting off and on, etc. During this period he lost track of time and often did not know where he was or who was with him. Since April 1 the patient had been sleepless, weak, nervous, excitable and despondent. His dizziness persisted, and he was unable to walk. He began to worry over finances and twice attempted suicide by turning on the gas.

Physical Examination on Admission.—There was slight anisocoria—the left pupil was larger than the right. There were rapid fatigability of the left external rectus, nystagmoid movement on turning the eyes to the right, tenderness over the masseters, some incoordination of the left leg as shown by heel-knee test, ataxic gait, swaying and falling in the Romberg position and coarse tremor of the extended hands. The spinal fluid was clear, containing 53 cells (mononuclear) and globulin + +. The Wassermann reaction was negative. The red blood cells numbered 4,820,000, the white blood cells 12,860, including: polymorphonuclear neutrophils 68 per cent., polymorphonuclear eosinophils 1 per cent., lymphocytes 24 per cent., large mononuclears 4 per cent., transitionals 3 per cent. The hemoglobin content was 78 per cent. The blood Wassermann reaction was negative. Urine examination was negative.

Mental Status on Admission.—The patient was restless, irritable and excitable, much disturbed and irritated by the slightest noise. He was correctly oriented. He said that he felt despondent and worried. His head felt "mixed up and not clear." Memory for recent and remote events was excellent. Retention and general information were good. He realized that he was physically and mentally sick.

Course in Hospital.—The temperature, which was 100 F. on admission, fluctuated between that point and normal until May 24, and then remained normal. It was with difficulty that the patient could be induced to leave his bed. He continued to be depressed, but gradually showed an increasing amount of activity. He was easily upset and went to bed on the slightest provocation. When refused constant attention, he threatened suicide. The slightest noise threw him into an irritable temper, and he would threaten to leave the hospital. When discharged, there was no evidence of any neurologic disorder except a fine tremor of the extended hands. The patient was transferred to a state hospital until recovery from the depression.

CASE 19.—*History.*—J. G., white, aged 38, married, a fireman, was admitted to the hospital, June 6, 1920, and discharged, July 28, 1920. One paternal uncle was insane. The patient had been an active, healthy boy with plenty of interests. He had no known sex difficulties. He had been a fireman for the past thirteen years.

During the first week of May, 1920, the patient had had a heavy cold with difficulty in breathing. He went to bed May 6, complaining of pain in the limbs, headache and nervousness. There was tremor of the legs. The patient slept little at first and began to talk wildly about fighting fires, pulled the furniture around to find the fire, saw moving pictures on the ceiling, etc. There had been marked irritability. At times the patient had difficulty in swallowing and there had been thickness of speech.

Physical Examination on Admission.—The pupils were equal and regular but eccentric. They were drawn upward and inward—the right more than the left. They reacted promptly to light and accommodation. There was no nystagmus or strabismus. The fundi were normal. There was a marked tremor of the lower jaw. Movements, however, were well performed. Speech was slurring and indistinct. The tongue protruded to the right. There was marked intention tremor of both hands. Deep and superficial reflexes were normal.

Examination of the urine was negative. The white blood cells numbered 6,800; the hemoglobin content was 92 per cent. The spinal fluid was clear, containing 22 cells (mononuclear) and globulin +. The Wassermann reaction was negative.

Mental Status on Admission.—The patient muttered to himself about fires. He was quiet most of the time but got up occasionally as if hunting for something. He was totally disoriented; he thought the physician was a lawyer and talked about being on trial for some street brawl. He spoke in a hazy fashion about being thrown out of bed and downstairs. His memory for remote events was good when his attention could be held. He showed some perseveration of words when asked to name objects, but his mind was so clouded that little stress could be laid on this. Recent memory and retention were poor. He wept during the examination, without apparent cause.

Course in Hospital.—The patient's mental condition gradually improved until by July 7 his mind was quite clear. Speech was still difficult and slightly stammering. There was some confusion of memory for his present illness, but otherwise he was quite clear.

Sept. 1, 1921: The patient complained of reversal of sleep curve and pains in the chest.

Dec. 28, 1921: The patient reported himself as entirely well.

Case 20.—*History.*—C. A. B., white, aged 36, married, a laborer, was admitted to the hospital, March 30, 1920, and died, June 17, 1921. His family history was negative. He had broad interests, was moderately alcoholic, friendly and sociable.

March 12, 1920, the patient became dizzy. There were slight (?) fever and pains in the elbows. During the first week he was very talkative and happy but said nothing unusual. On March 19 he did not seem to have much life, was blue and kept to himself. On March 20, he began to tremble violently, although he was not cold. Sleep became poor, and he paced the floor at night. His depression became more pronounced. The attacks of tremor continued, and on the 29th his speech became thick and he had difficulty in swallowing. There was also diplopia.

Physical Examination on Admission.—The patient complained of "numbness in arms and legs, pain around the heart and smothering sensations." His temperature was 100 F. His pupils were regular—the left larger; reaction to light and accommodation was slight. There was lateral nystagums on looking to the left, but no strabismus. The eyegrounds were clear. Facial and jaw movements showed some weakness, but there was no inequality of the two sides. The patient was unable to swallow and speech was thick and weak. There was coarse tremor of the tongue, face and hands, and at times a coarser tremor involving the legs and arms. The deep reflexes were active but equal. The patient perspired profusely, pools of fluid collecting in the ears. The face was masklike. There was marked general weakness and some rigidity. The patient held positions when put in them. No disturbance of sensibility was present. Examination of the urine was negative. The white blood cells numbered 12,050. The spinal fluid was clear, containing 19 cells (mononuclear); globulin + +. The Wassermann reaction was negative in the blood and spinal fluid. The colloidal gold curve was 2322100000.

Mental Status.—The patient was correctly oriented, quite cooperative and interested in his examination, but depressed and anxious about his condition. He said that he felt as if he were dying and called out for help.

Course in Hospital.—The patient became more stuporous, the rigidity increased, and the tremor of the body became almost constant. On April 11 respiration became labored (32), and there was right facial weakness. His temperature remained around 100 F., the pulse reached 120. The Babinski and Kernig signs were negative. The urine showed some granular casts and albumin + +. The phenolsulphonephthalein was 50 per cent. in two hours. The white blood cells numbered 13,960.

At times he recognized those about him. His talk was muttering and indistinguishable, but chance words showed that there was some clouding.

On April 4, the patient's condition had improved slightly, but there was slight internal strabismus on the right and some left facial weakness. The following day the patient became comatose and incontinent and showed tremor of the eyeballs. The spinal fluid showed 40 cells and globulin + +, and a colloidal gold curve of 3443532111. His condition became slightly improved, and he again recognized those about him. His temperature remained about 101 F. The neurologic status showed no change. The leukocytosis on April 6 was 14,140. On April 12 there was a Kernig sign on the right side; knee and ankle reflexes were more active on the left. The upper abdominal reflexes were absent. The pupils were dilated and fixed. The rigidity was profound. The patient was, however, much more alert. He did not talk but obeyed commands and answered questions by opening and closing his eyes. On April 14, there was retention, and the patient had to be catheterized. The temperature was still elevated. On April 22 the general condition had improved, although fever, rigidity, tremor and the other neurologic signs persisted. By April 29 the patient was afebrile, but otherwise there was no change except left-sided ptosis. The white blood cells numbered 18,000. May 1 he developed pulmonary edema—respiration 48, pulse 146 and temperature 101.4 F. He was given strophanthin and epinephrin intramuscularly. The temperature rose to 103 F. He soon began to improve and by the morning of the 6th was in the same condition as before the pulmonary edema, but decubitus ulcers began to develop. On May 27, the patient spoke for the first time, saying, "It is about time to get well or die." At the same time he referred to some event that had happened a week before and tried to help put on his nightshirt. The rigidity and neurologic status continued. On May 15 the spinal fluid was normal.

On June 17, the patient's attitude was still fixed and rigid. He could perform simple, spoken commands. The pupils reacted briskly to light but poorly to accommodation. The left pupil was slightly larger and the eye movements jerky. There was tonic contraction of the jaw muscles; the head was slightly retracted and the neck stiff. The arms were adducted, semiflexed and pronated and the finger semiflexed with thumbs opposed and adducted. Full extension of the arms was restricted by contracture. There had been little change except that he seemed to take in what was going on around him. He said that his bedsore was less painful, and that the man in the next room was to be operated on (correct).

Case 21.—*History.*—H. G., white, aged 17, single, a boilermaker, was admitted to the hospital, May 21, 1920, and died, May 30, 1920. His family history was negative. He had had influenza, October, 1918.

May 13, he complained of pain at the vertex. The next day this extended down the sides of his neck, and later into the throat and ears. On May 17, he complained of feeling nervous. The same night he was restless, sleepless and talked incessantly. Since onset he had not slept and could not be put to sleep even with morphia. He complained of blurring of vision and difficulty in swallowing. His talk gradually became incoherent and confused.

Physical Examination on Admission.—The patient complained of soreness of the head, neck, arm and epigastrium. The temperature was 101.2 F. The pupils and eye muscles were normal; there was no nystagmus. There was left ptosis. The speech was thick and careless and the voice hoarse and weak. There was no disturbance of sensibility. Deep and superficial reflexes were equal and active. There was marked tremor of the extended hands. The gait was unsteady and the Romberg sign positive. The urine showed acetone bodies and some albumin.

The white blood cells numbered 6,800; the hemoglobin content was 90 per cent. The spinal fluid was clear, containing 16 cells (mononuclear), globulin +, with a colloidal gold curve of 0001111000. The blood and spinal fluid Wassermann reactions were negative. The blood pressure was: systolic 180, diastolic 120. The heart was enlarged to the left.

Mental Status on Admission.—The patient was in constant motion, writhing about, sitting up, staggering about and complaining of great pain. He talked continually, calling his mother and sister, and constantly went through the movements of his occupation. When his attention could be held he answered relevantly, but his talk tended to drift off. Throughout he said that he was in good spirits, although crying out with pain. He was only partially oriented —he knew he was talking to the physician but thought he was at home. It was impossible to test memory, retention and calculation on account of poor attention.

Course in Hospital.—The patient's temperature remained at 102 F. until death. Although the eyes were closed most of the time, it was impossible to get him to sleep. On the 23d, he became more restless, complained of pain in the abdomen, hips and elbows, appeared stuporous and began to hiccup. On the 24th he had twitching of the abdominal muscles. On the 25th the delirium had become more pronounced, but his stupor also increased. The respiration became irregular, and the hiccuping increased. On the 26th bilateral ptosis was observed. He died on the 30th with respiratory failure.

CASE 22.—*History.*—J. L. R., white, aged 63, a retired sea captain, was admitted to the hospital, April 15, 1920, and discharged, April 21, 1920. His family history was negative. He had general good health. He was good-natured, cheerful and social, but high-tempered. During July, 1919, there was a noticeable let-down in the patient's activity, endurance and spirits. In October, 1919, an automobile accident caused a brief period of unconsciousness, but no after-effects were noted except some nervousness and timidity.

In January, 1920, the patient began to sleep poorly and was very restless. January 14, he showed a sudden change, becoming drowsy and listless and he complained of seeing double. There was no improvement in February, and for ten days he had "mild convulsions" with twitchings, especially about the face and mouth. His tongue swelled (?), and speech was thick. At night he was mildly delirious. After the last of February he began to improve, but he felt very weak, was easily fatigued and his memory was poor, especially for recent events.

Physical Examination.—The patient complained of weakness and numbness in his right leg from his hip to his knee. The temperature was normal. The right pupil was larger and irregular; pupils reacted promptly to light and accommodation. There was no diplopia, nystagmus or strabismus. The eyegrounds were clear. There was no evidence of arteriosclerosis. There was no weakness of the facial or jaw musculature. Speech was thick. No evidence of sensibility disturbance was present. Deep and superficial reflexes were equal and active. A slight tremor of the extended hands was present. The heart was not enlarged, and the peripheral vessels were not sclerosed. The blood pressure was systolic 118, diastolic 70. The urine was normal except for the presence of hyaline casts. Phenolsulphonephthalein excretion was 58 per cent. in two hours. The white blood cells numbered 7,500. The hemoglobin content was 75 per cent. The red blood cells numbered 4,032,000. A roentgenogram of the head was negative. The spinal fluid was clear, under no increase in pressure, and contained no cells; globulin + +; colloidal gold curve 4433211000. The Wassermann reaction on the blood and spinal fluid was negative.

Mental Status.—The patient was listless, moving little and then very slowly. His attention was good, but he was very solemn and did not smile. There was no spontaneous talking but he answered questions relevantly, promptly and with normal inflection. He said he was not depressed, but complained of some difficulty in thinking. Memory for remote events was slightly impaired; retention was poor. He could not remember a test address after five minutes and could repeat only five digits. He retold a test story, however, very well. Calculation was good. Writing was tremulous and uneven.

Course in Hospital.—During the patient's stay in the hospital for one week, there was no change in his condition. Repeated urine examination failed to show any more casts.

Case 23.—*History.*—D. B., white, aged 38, married, a housewife, was admitted to the hospital, July 6, 1920, and discharged July 12, 1920. Her family history was negative. She had had headaches all her life. There had been progressive increase in weight since the birth of the first child. She had had a severe attack of influenza in the fall of 1918, since which time she had not felt as strong.

The last week in March, 1920, the patient had nocturnal earache and immediately afterward had an attack of generalized muscular twitching without loss of consciousness. Several more such attacks followed in the ensuing ten days. During the last week in April, bilateral ptosis developed with dimness of vision and diplopia. The patient improved gradually but complained a great deal of rectal tenesmus. While at another hospital, she is said to have had some fever and her systolic blood pressure was found to be 200. Preceding her admission to this hospital she had been sleepless, depressed, listless, and had had aches and pains in "stomach and limbs." For the week preceding admission, there was some difficulty in swallowing.

Physical Examination on Admission.—The patient was obese, with large pads of fat which had a doughy feel. The skin was coarse and dry, and the hair of the head and eyebrows was scanty. The temperature was 100.8 F. There was a bilateral ptosis, more marked on the left. The right pupil was slightly larger, both slightly ectopic, displaced upward and inward. Both reacted promptly to light but sluggishly to accommodation. No diplopia, nystagmus or strabismus was present. Both disks were clear, the veins injected, the arteries tortuous and a little thickened. There was no facial or jaw muscle weakness. Deep and superficial reflexes were active. There was no tremor. Gait, speech and writing were normal. Sensibility was not disturbed. The urine was clear. Blood pressure was: systolic 162, diastolic 110. The heart was not enlarged. The white blood vessels numbered 8,900. The hemoglobin content was 95 per cent. The spinal fluid was clear, containing 6 cells (mononuclear), globulin +. The Wassermann reaction was negative, colloidal gold curve 2445221100. The blood Wassermann reaction was also negative.

Mental Status on Admission.—The patient lay in bed with eyes half closed, giving the impression of greater dulness than her talk indicated. Her spontaneous talk was concerned mainly with her somatic complaints, which were chiefly rectal pain and indigestion. She was not oriented correctly for date, but recognized place and person. She said she felt sad because she had to be sick, with five children at home. At times she was jocular and made rather poor jokes. Her memory for remote events was excellent, and, although she recalled isolated occurrences of the preceding twenty-four hours, she could not give a consecutive account. She was unable to repeat more than six digits. She showed good grasp in a test story. Her general information was fair, but the tests showed inattention. She realized that there was something wrong with her mind.

Course in Hospital.—The patient's condition remained practically unchanged during her six days' stay in the hospital. She was very hypochondriacal and at times restless and slightly confused. She would get out of bed to look for a cathartic after the nurse had told her she was bringing her one. On one occasion she thought the nurses were talking with other patients about her. She showed slight elevation of temperature throughout, up to 99 F.[11]

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[11] In addition to the references given, the following may be of interest:

Bassoe, P.: The Delirious and the Meningoradicular Types of Epidemic Encephalitis, J. A. M. A. **74**:1009, 1920.

MacNalty, A. S.: Report of an Inquiry Into an Obscure Disease, Encephalitis Lethargica, Rept. Local Govt. Board on Pub. Health and Med. Subjects, N. S., No. 121, London, 1918, pp. 12-69; also Proc. Royal Soc. Med., London **12**: 1918 (1), Sect. Med. 1-23.

Maier, H. W.: Epidemic Encephalitis at Zurich, Schweiz. med. Wchnschr. **1**:249, 1920.

Tilney, F., and Riley, H. A.: Epidemic Encephalitis with a Consideration of Some of Its Prominent Clinical and Pathological Manifestations, Neurol. Bull. **2**:106-135, 1919.

Kirby, G. H., and Davis, T. K.: Psychiatric Aspects of Epidemic Encephalitis, Arch. Neurol. & Psychiat. **5**:491 (May) 1921.

Abstracts from Current Literature

A STUDY OF NISSL'S STAEBCHENZELLEN IN THE CEREBRAL CORTEX OF GENERAL PARALYSIS, SENILE DEMENTIA, EPILEPSY, GLIOMA, TUBERCULOUS MENINGITIS AND DELIRIUM TREMENS. U. N. Noda, J. Nerv. & Ment. Dis. **53**:3 (March) 1921.

The author takes up the descriptive histology of the "staebchen" cell, reviews the literature and gives in detail his own studies on selected groups of cases. As to the morphology, the "staebchen" cell is a rod-shaped cell with a markedly elongated nucleus, the nucleus staining faintly and containing one or more nuclei. Threadlike substances going out from the poles are frequently the only evidences of protoplasm. The nuclei may vary a good deal as to shape and yet retain the elongated appearance. It is found in the cerebral cortex in various diseases and especially in general paralysis in which it constitutes the most important histologic finding.

The question of origin of this cell is discussed at length in a careful review of all the contributions on the subject. Nissl believed that they were found parallel with the blood vessels for the most part, but if numerous were likely to be irregular in distribution. Alzheimer substantiated the frequency of "staebchen" cells in paresis and the parallelism to the vessels in the cortex, but added that they were irregular in the deeper layers. He stated that they originated from glia. He held to the view of an adventitial origin as they were found around blood vessels in the adventitial spaces and resembled adventitial cells in so many ways. Cerletti advocated a glial origin but believed they were a part of an otherwise invisible vessel. Straeussler agreed with the view of a glial origin, claiming to have found glial fibers given off and present in large numbers wherever glia cells were numerous. Ris found these cells indistinguishable from adventitial cells of the vessel walls, though believing in a glial origin. DeRuck stated his belief in a fibroblastic origin. Mott wrote that they were "probably collapsed capillaries." Ranke advocated a glial origin. Dupre found them largely in the frontal and central convolutions in general paralysis, more numerous in the large pyramidal layers with a parallelism with the axis cylinders of the nerves, and thought they were due to the paretic condition. Persini believed that they originated from glia. Rosenthal affirmed an adventitial origin. Bonfiglio accepted both glial and adventitial origins. Rondini concluded that they were abnormal products resulting from a progressive process in the vessel wall. Ulrich found the greatest number in general paralysis, in the precentral convolution in the polymorphous layer, vertical to the surface, and with a definite relationship to the vessel alterations. In meningitis she found them in the molecular layer under the pia, stating that they are significant in the histopathologic diagnosis of general paralysis and cerebral syphilis, important in meningitis and multiple sclerosis, but in other diseases of little significance. She inclined to a mesodermal origin. Lotman found them in seven cases of glioma. Achucarro, in a study of rabies, found them in the stratum radiatum and explained that this condition was due to the structure of this region. He believed that they resulted from degenerative processes but might develop in part from ectoderm and in part from mesoderm. Sinchowicz stated that they were more frequent in those cases of senile dementia which ran a rapid course and believed that they arose from glia. Bassoe and Hassin found them in epidemic encephalitis, especially near the infiltrated vessels in the pia and the cortex.

The author studied several groups of cases from the standpoint of prevalence and charateristics of the "staebchen" cells. He gives the histories and pathologic findings in detail. In the general paralysis group the general histopathologic picture given is that usually accepted and typical of the disease. He regarded all rod-shaped cells as "staebchen" cells and found them in large numbers, sometimes as transition forms from glia cells. They were particularly numerous in the frontal region and cornu ammonis, but fewer in the temporal and occipital lobes and cerebellum. In the white matter they were fewer than in the gray in the cerebrum, but in the cerebellum the reverse order obtained. In the cerebral cortex the greatest numbers were present in the middle laminae, especially in the small and large pyramidal layers—sometimes there were ten or more in a single microscopic field. The long axis of the "staebchen" cell in the cerebellum was vertical to the surface in the molecular layer, but parallel to the radiations in the marrow. In the cerebrum they were parallel with the radiations only in the deeper portions. Elsewhere they were irregularly placed. In the cortex the general direction was vertical and parallel with the long axis of the nerve cells, especially in the small and large pyramidal layers. No relation in the number of "staebchenzellen" and extent of nerve cell degeneration was found. Rod-shaped trabant cells were numerous wherever there was an increase in trabant cells. There was, however, a parallelism between the "staebchen" cells and glia proliferation. Various transitional stages between glia cells and "staebchen" cells and spider cells with elongated nuclei were found. "Staebchen" cells were found in colony-like formations. In regard to the relationship with blood vessels, there was a parallelism between the number of "staebchen" cells and the extent of perivascular infiltration. Sometimes elongated adventitial cells resembled "staebchen" cells (but were probably regressive capillary changes), and the author believes it may be the origin of some of them. Protoplasmic elongations from the poles were characteristic of these cells. The nuclei were from 15 to 30 microns in length. Pigment granules seen in some were of a brownish or yellowish color and situated mostly at the poles. Many were also found loaded with fat granules. The cells as a whole were well stained with toluidin blue, thionin and the Weigert stain. Many spider-like types of glia cells were seen with elongated nuclei, which the author believes should be called "staebchen-like" spider cells.

In the senile dementia group the usual arteriosclerotic changes were found generally. In addition, however, there was a swelling and splitting of the elastic lamina. Glia changes were both progressive and regressive. Some spider-like cells showed elongated nuclei. "Staebchen" cells were not numerous, perhaps two in each field, most common in the cornu ammonis, the white substance of the cerebellum and the molecular layer of the cerebrum. The direction of the cells was the same as in the general paralysis group, but the nuclei were shorter and there was less protoplasm than in paresis, and more frequently regressive changes were found. In the senile plaques the author found glia cells taking part, some having rod-like nuclei, but they were not definitely "staebchen" cells. There were some rodlike spider types of glia cells.

In the epilepsy group there was a chronic cell alteration, proliferation and regressive changes in the glial elements. "Staebchen" cells were few—one in a single field—with the greatest number in the cornu ammonis. In the delirium tremens group there was chromatolysis, alteration and pigmentation of nerve cells. There was an increase in glia cells, some of them bearing fat pigment, as did the nerve cells. "Staebchen" cells were few in number, short in length, lacking in protoplasmic processes, the majority being loaded with fat. In tuberculous meningitis these cells were more numerous than in senile dementia but less than in general paralysis. The outer cortical layers and those places in which infiltration of the blood vessel walls was most pronounced contained the largest number of "staebchen" cells. They were also found in the pia. The morphologic characteristics were essentially the same as in the other diseases described, but the nuclei were shorter, and the protoplasmic processes were present. Some held pigment. In glioma the "staebchen" cells had more cytoplasm, wider nuclei and protoplasmic processes that were shorter than in general paralysis. They were more numerous in the tumor mass than elsewhere.

The author's conclusions are that "staebchen" cells are the most important histopathologic finding in general paralysis; that they appear in appreciable numbers in meningitis and are fewer in other diseases, with the exception of glioma. There are typical and transformation forms. He classifies them as: (1) those having elongated nuclei with protoplasmic processes, most frequently found in general paralysis; (2) those with short nuclei and scant protoplasmic processes, seen in glial proliferation and more common in senile dementia; (3) all curious and bizarre shapes; (4) transitional forms between "staebchen" cells and glia cells, occurring when glial proliferation is marked and including the "staebchen-like" spider (glia) cells. In general they parallel the surrounding elements and originate partly from ectodermic and partly from mesodermic tissue. In a discussion as to the theories of the causes of glia cells becoming "staebchen" cells, he considers a mechanical factor—rodlike forms produced by adaptation to available spaces, the results of osmotic pressure, and lastly the supposition that the special function which glia cells are called on to perform can best be accomplished in an elongated shape.

Patten, Philadelphia.

EPIDEMIC ENCEPHALITIS. K. Bonhoeffer, Deutsch. med. Wchnschr. **47**:229 (March 3) 1921.

Bonhoeffer sums up his experience with epidemic encephalitis as follows:

1. Prodromal Period: Headache is one of the first complaints. This is usually less intense than headache occurring in meningitis. It is most frequently localized in the posterior fossa and has a tendency to radiate to the neck and shoulders. Fatigue and dizziness are common. Temperature is usually present, but it is of low grade. Bronchitis or intestinal disturbances were encountered in only three of twenty-seven of his cases. Visual disturbances occur among the early symptoms in the majority of cases. Tinnitus and paresthesia in the distribution of the fifth and other nerves are not infrequent. The occurrence of anxiety and even delirium during the first few days is characteristic. A change of facial expression is usually noted at the onset. Insomnia or lethargy may occur. The prodromal period may last for several weeks.

2. Types: Bonhoeffer divides his cases into three main types: (*a*) lethargic; (b) parkinsonian, and (*c*) choreiform. He mentions the myoclonic forms but does not group them.

Among the ocular palsies, paralysis of accommodation is most common, next ptosis, and finally conjugate deviation. Bonhoeffer noted loss of light reflex or diminution to light and accommodation in a considerable number of cases. In a few cases paresis of the tongue occurred, with difficulty in swallowing. The sensory fifth nerve is seldom involved, but occasionally motor weakness is noted. Lethargy and insomnia are encountered rather constantly. Temperature is usually slight and of long duration. High continued temperature is usually looked on as indicative of complications.

Nearly all patients showed increase of globulin and a lymphocytosis in the spinal fluid. Hemiplegic symptoms of pyramidal tract character and cortical affections were not found in the author's series. The Babinski sign or ankle clonus was rarely noted; there was more frequently diminution or loss of the Achilles' reflex. Vasomotor disturbances, retention of urine and constipation were not infrequent.

These are the usual symptoms. Next in frequency is the choreo-athetoid type. Bonhoeffer does not believe in considering these two types as sharply divided from one another, but calls attention to the fact that both are extrapyramidal motor disturbances. There are cases, however, in which marked choreiform movements are found from the onset, and which from the pathologic findings must be considered as thalamic and subthalamic in character.

3. Diagnosis: The typical case offers no difficulty in diagnosis. Bonhoeffer states that with his increasing experience many of the old nondiagnosed cases have become clear. Thus, a case of acute paralysis agitans, as also a case resembling Huntington's disease which he saw several years ago, now seem clear to him.

4. Differential Diagnosis: Cerebral syphilis, general paralysis, catatonia, myasthenia and chorea must be considered in the long standing atypical cases. Syphilis at times is especially difficult to rule out, as Bonhoeffer has noted the occurrence of a positive spinal fluid Wassermann reaction in encephalitis. The value of stiff pupils has also been impaired by their occurrence in encephalitis. Chorea is at times difficult to diagnose as the more severe types are not uncommonly end stages of encephalitis.

Frequently the diagnosis can only be made because of the prevalence of encephalitis. The author mentions the case of a patient who complained of pain in the back of his neck with marked fatigue. Because of the epidemic, a spinal puncture was performed which showed increase of globulin and lymphocytosis, thus indicating the diagnosis.

A cerebellar syndrome is also recognized by the writer. In one case tremor, headache and delirium occurred; later marked static disturbances developed with nystagmus which gradually disappeared. The spinal fluid in this case also showed a lymphocytosis and increase of globulin.

Hiccup was observed in one case. Two patients had acute atrophy of the tongue, nuclear in type. The possibility of a localized acute poliomyelitis in the medulla was considered. Bonhoeffer does not believe that poliomyelitis and encephalitis have the same identity, as in the latter, nuclear lesions tend to recover.

Finally, there is another group which under the picture of a dissociation and delirium progress rapidly to death. In some of these cases one must satisfy himself with a diagnosis of acute delirium.

5. Course of Disease: The course of the disease is variable. The acute cases in which death occurs in a few days are rare. The illness usually lasts

from four to eight weeks or longer. The lethargy has continued for months in some cases. Marked variation in symptoms is not infrequent. Thus the rigid type may develop choreiform movements, the lethargic type, delirium, etc.

Patients with cranial nerve palsies show a distinct tendency to recover. Difficulty in accommodation is the symptom that has a tendency to remain longest. More obstinate are the disturbances of extrapyramidal motility. Pictures of Huntington's chorea and paralysis agitans have been observed to continue for more than a year. Bonhoeffer has also observed long continued psychotic manifestations, especially types resembling cocainism.

Paresthesias of the head and trunk, emotional weakness and hypochondriacal ideas are frequent residuals. Amnesic disturbances were not noted, and Bonhoeffer states that he has not seen them recorded.

Intellectual defect was not noted in any case. This is important as frequently the expressionless face makes one think it present.

6. Prognosis: The prognosis in Bonhoeffer's series does not correspond to that of the Vienna Clinic in which one third of the patients died. Bonhoeffer states that unless complicated by pneumonia or other troubles, the prognosis is not unfavorable. It is bad in cases of severe delirium or deep stupor with high temperature. This type, however, is rare as compared with the milder and chronic forms. As a rule, one may say that patients who survive the first few weeks will recover. Occasionally some complicating factor may develop, as in one of the author's cases in which paralysis of deglutition led to bronchial pneumonia and death.

The tendency to the development of pulmonary complications is well known. The majority of patients who came to necropsy died of pneumonia. The theory that these patients had some involvement of the dorsal vagus nucleus is worthy of consideration, but requires further anatomic proof. The tendency for relapses to occur is not unusual, but as a rule succeeding attacks are milder.

7. Pathology: It is the opinion in Bonhoeffer's clinic that encephalitis is a disease of fairly characteristic localization. Macroscopically, usually nothing is to be noted. In one case miliary hemorrhages occurred. Microscopically, a lymphocytic infiltration of the blood vessels occurs. Small hemorrhages and foci of active glia proliferation and cell destruction are also present. The gray matter of the pons is especially involved, but the cortex, cord and pia show infiltration. Large foci do not occur as a rule.

Restitution of destroyed areas is a characteristic finding. Judging by the general clinical and pathologic picture, there is little doubt that much of the damage is toxic in nature. At the present time it is impossible to separate the types of encephalitis from an anatomic standpoint.

8. Etiology: No etiologic factor is as yet known. Bonhoeffer was able in several cases to isolate almost in pure culture pleomorphous streptococci described by Economo, but the presence of this organism in other infectious processes causes the author to hesitate to consider it the etiologic factor. Inoculation of the spinal fluid and brain matter have proved of no value. The consensus of opinion appears to be, according to Bonhoeffer, that encephalitis is a localization of the "grip" infection in the brain.

9. Therapy: The author concludes his paper without offering any special therapeutic measures.

Moersch, Rochester, Minn.

SPEECH, ITS DEVELOPMENT AND INTEGRATION WITH THE INTELLIGENCE. Michael Osnato, M.D., Neurol. Bull. **3**:47 (Feb.) 1921.

Osnato's paper is a plausible attack on one of the bulwarks of clinical neurology—the diagrammatic conception of speech. He distinguishes sharply between articulate speech which is merely a kinetic act involving the whole body and speech in a broader sense which is an intelligent learned function dependent on the acquirement and use of sense symbols, sound, visual, gesture (kinesthetic and proprioceptive), olfactory, and tactile. Even in parrots and primates the ability to utter speech is correlated with other evidences of intelligence. Garner in his popular treatise describes nine monophrastic expressions, one phonetic sound, and at least thirty "words" used by the higher apes to express elemental emotions and communicate with each other. The level of this intelligence is in doubt. Kinnaman regards it as "intermediate abstraction" which is a more complicated form of mental process than the simple idea or notion or the mere memory of a particular sensory perception. The human animal develops intelligent speech through a series of evolutionary steps beginning with the baby or monkey or parrot stage and reaching its ultimate differentiated state which is the production and expression by means of spoken language of abstract thoughts and concepts. The latter acquirement is solely restricted to the human adult. The infant utters unintelligible sounds and learns by repetition of results or reactions in the parent to associate the sound with the fulfillment of a desire. For instance, "goo-goo" may be interpreted by the mother as a call for water and the infant then associates water and the satisfaction which this gives to its needs with the production of the sound which it has made. The pathways which determine speech are sound, visual, gesture (kinesthetic and proprioceptive), olfactory and tactile, and in some sense their exercise and development is individual and uneven and somewhat governed by environmental factors.

Turning to anatomopathology Osnato finds another weapon to use against the "brain pattern" idea. He says "the only association fiber pathways which have any definite degree of constancy and which can only be made out in the gross by anatomical dissection after hardening of the brain in appropriate fluids, are the cingulum, the fasiculi-longitudinalis inferiores and superiores and the arcuate fibers. Various other association bundles are known, but are inconstantly arranged and not always easily identified. Further, the author believes that clinicopathologic correlations are generally wanting and rather indicate that the adherents of the "brain-spot-aphasia" theory can only see the spot and not the remainder of the brain. He quotes Mautier (L'Aphasie de Broca, Paris, 1908, Vols. 1 and 2) to advantage. In fifty-seven cases in which the patients during life presented clinical evidence of aphasia, the foot of the third left frontal convolution was absolutely intact; in twenty-seven in which aphasia had been absent it was destroyed; and in only eight of 103 necropsy examinations was Broca's dictum borne out. Marie concludes that "there is not one case of tumor involving exclusively the foot of the left third frontal convolution in right-handed individuals which has ever caused aphasia. Many times this convolution has been completely destroyed by the tumor and no aphasia has resulted." Frankel and Onuf's statistics are also somewhat confirmatory (Frankel, J., and Onuf, B.: Corticale and subcorticale motorische Aphasie und deren Verhaltnisse zur Dysarthrie, Deutsch. Ztschr. F. Nervenheilk. **15**:312, 1899). Burchhardt's daring attempt to produce aphasia in two noisy, overproductive insane patients by brain operation is interesting. "He removed 5 grams of the cortical gray from the foot of the first and second left temporal gyri and no word deafness resulted. Eight months later in the same patient he resected the top and foot of the left third frontal convolution and no agraphia or motor aphasia resulted. In another patient this surgeon removed a portion of the left supramarginal convolution and destroyed a considerable portion of Wernicke's sensory speech area and no aphasia resulted. In this last patient the left third frontal convolution was also resected. The patients continued just as voluble and full of noisy speech as before." Osnato asserts that "it is our belief that in a much greater number of patients the process of the acquirement of speech and intelligence has been so irregularly different and individualized that to talk of patterns and any longer to hold the diagrammatic conception of speech as a function localized in definite centers is impossible and unscientific and is in contradiction of all our anatomical and pathological data and our knowledge of the development of speech in children and articulate utterance in intelligent animals."

In general, Osnato's conclusions are not in excess of his premises. Several of the premises may be and have been less definitely interpreted. For instance, certain investigators would probably be unwilling to concede to primates much beyond mere memory for sensory perception. Again, additional clinicopathologic material might counterbalance the striking array which the author presents. Nevertheless, the conception of a pathologic brain spot as a sole determinant of clinical aphasia rests on a less sure foundation at present than it did in the past.

STRECKER, Philadelphia.

SENSORY APHASIA WITH REMARKS ON THE PATHOLOGY OF MEMORY. WALTER VON SCHUCKMANN, Monatschr. f. Psychiat. u. Neurol. **48**:232 (Nov.) 1920.

The patient, a man, aged 29, had a stroke in February, 1919, which resulted in a left-sided paresis. June, 1919, a second stroke was followed by loss of speech. The patient was right-handed. Eight days after the stroke, his wife noted that the patient did not understand what was said to him. He became irritable and violent and was admitted to the hospital, July, 1919. He died, December, 1919.

Necropsy revealed:

1. Atrophy of the posterior third of the first left temporal convolution and adjacent areas, reaching up to the angular gyrus.
2. A medullary cyst, the size of a plum, lying beneath the supramarginal gyrus.
3. Other slight pathologic changes which would seem to have no relation to the aphasia.

Clinically, the most striking feature was the patient's inability to express himself in speech. Spontaneous speech consisted of a mass of meaningless and confused syllables. In contrast to this, word-finding for concrete objects was relatively good. The results of his responses in the naming of concrete objects are:

	Percentage
Understanding of spoken words	90.0
Speaking from dictation	70.0
Spontaneous word finding	65.0
Reading aloud	22.2
Understanding of written words	60.0
Spontaneous writing	0.0
Copying	0.0

There is a striking difference between reading and writing. The percentage of reading can be explained on the basis of a sensory aphasia (first temporal convolution) plus some involvement of the angular gyrus. The complete loss of writing must have a different source. The author considers it a subcortical agraphia, due to the interruption of fibers from the writing center to the hand center by the subcortical cyst which lay beneath the supramarginal gyrus.

The author calls attention to the fact noted by Bastian and illustrated by this patient, that in partial lesions of speech centers, speech reactions are produced more easily by objects than by ideas. In this patient 65 per cent. of the attempts at word-finding for objects seen or felt succeeded, but word finding through association or ideation was nil. This the author tries to correlate with Liepmann's findings. Liepmann finds that the left hemisphere has a predominating influence in praxia; the left hemisphere alone can compass purposive movements without the object, whereas the right hemisphere is quite able to bring about purposive movements with objects. Similarly the author believes it possible that the right hemisphere through the right-sided sensory speech zone is able to produce a word-sound picture in the presence of the optic or tactile impressions, whereas only the left sensory speech zone can bring up the word sound picture through association or ideational paths.

The author then proceeds to a discussion of certain features of the pathology of memory, basing his remarks on the work of Semon. Semon speaks of the general characteristics shown by living organisms of being permanently impressed and changed by a single stimulus, as its engraphic receptivity. The permanent change produced as the engraphic effect of the stimulus is called an engram. The engram has a latent tendency to recur. The activation of this latent tendency he speaks of as ekphoria. An ekphoric effect on the engram may be produced by a return of the original stimulus or by a stimulus which has produced a related or associated engram. The ekphoric effect produced by the return of the original stimulus is greater than that produced by a merely associated stimulus (or stimulus producing an associated engram). The author points out that milder grades of disturbance of the engram will do away with its reproduction through associated stimuli, but will leave undamaged its ekphorability (ekphorierbärkeit) through a return of the original stimulus. This "hypesthesia" of the engram may explain certain features of aphasias. On recovery from sensory aphasia, speaking on dictation improves more rapidly than spontaneous speech. One generally explains this by saying that speaking on dictation is more resistant. A more plausible explanation is a partial disturbance of ekphorability. At the heights of the disease the ekphorability of the word sound engram is lost both for the original and for associated stimuli (anesthesia of the engram). As the disturbance clears, the ekphorability by means of the original stimulus returns, while that for associated stimuli remains lost. The original stimulus for word sound engrams is the heard word; and the test for the ekphoric effect of the original stimulus is repeating the word on dictation. Therefore, speaking on dictation returns while spontaneous speech remains disturbed. On the same basis the author attempts to explain the fact that in recovery from sensory aphasia understanding of spoken words recovers more quickly than speech. He then applies this same law to the case reported, explaining the slight involvement of understanding of spoken words in contrast to the marked disturbance of spontaneous speech by this same hypesthesia of the engram. This is offered as a possible substitute for his previous explanation, namely, the intervention of the right sensory speech field.

SELLING, Portland, Ore.

CLINICAL ENDOCRINOLOGY. Walter Timme, Neurol. Bull. **3**:3 (Jan.) 1921.

Timme introduces his comprehensive point of view of the internal secretory glandular system by dividing mankind endocrinologically speaking into three epochal life periods. The first, presided over by the thymus and pineal glands, embraces the span of years from birth to puberty (accretion, growth, and development) ; the second, under the domain of the gonads with control exerted both here and in the initial stage by the thyroid, pituitary and suprarenal glands, includes the period of life from puberty to prime (advance, aggression, and reproduction) and finally, gradual deterioration to dissolution (involution) marked by diminution in the activity of the last mentoined triad of glands.

The thymus is credited with the power of favoring somatic growth with an inhibitory influence on the sex glands. The clinical characteristics of thymic subinvolution, which is interpreted as an extension of glandular functioning beyond the time of ordinary involution are: "peaches and cream" complexion, absence of beard, mustache, and sparse or feminine type of pubic hair, bluish white teeth, large central and small lateral incisors with cutting edge instead of fang canines, flail-like joints, "torus palatinus" and delayed epiphysial-shaft union. In addition, there is "low blood pressure, slow pulse, hyperacidity, fatigability and usually subnormal temperature. The genital apparatus is usually small, but besides this it also shows some lack of differentiation, so that the penis emerges from a scrotal fold which may entirely surround its base." This seemingly clear picture is greatly obscured by the intrusion of the compensatory sympathicotonic mechanism of the suprarenals, pituitary and thyroid glands. There is in the classic thymic patient a psychic syndrome of childishness, imitativeness, self-centering, obstinacy, negativism, and in general an inability to bear the brunt of the struggle for existence. The converse of this, or precocious involution, produces contrasting physical and mental earmarks: somatic precocity in teeth, bones and hair; and psychically, initiative, easily aroused anger and resentfulness. There is "mature-precociousness" if one may use such an indefinite term.

Pineal subinvolution is supposed to determine retarded sexual development, "such retardation being of a direct growth character without the concomitant of reversion or lack of differentiation." There is also unusual muscular development. The reverse or premature pineal atrophy reveals radiographic "sand" before the twentieth year, indications of muscular insufficiency and relative genital and psychic overdevelopment.

There follows an interesting discussion of the thyroid-suprarenal-pituitary group. To quote the author, it is "this group that controls quantitatively and periodically the activity of the other internal secretory organs and the intensity, regularity and reactive capacity of the functoning of the vegetative nervous system. In short, it controls the automaticity of the individual in his adjustment to environment, in his reaction to fatigue, disease, to anger and other emotions, to competition in the struggle for existence, and prepares the organism physically to meet the shocks attendant on it." (One may fairly hesitate before this all-embracing claim.) In discussing the underactivity of the thyroid, nothing new is brought to light. Emphasis is placed on the numerous intermediate stages which may exist between frank cretinism or myxedema and normality. The anterior lobe of the pituitary gland often compensates for thyroid insufficiency. The understanding of certain of the more indefinite hyperthyroid reactions becomes easier, if we can assume with Timme that the gonads and suprarenal cortex are directly asynergic with the thyroid and that on them depends the maintenance or loss of thyroid balance.

Because it rests on a more secure foundation it may be well to repeat the sympathicotonic syndrome of plus suprarenal functioning: (1) delayed peristalsis, (2) rapidity of pulse, (3) flushing of the skin, (4) lessening of the secretions generally, (5) mobile and frequently large pupils, (6) deficiency in the gastric juice and in its acidity, (7) heightened temperature, (8) a heightened blood pressure and a general increase of metabolism." Mentally we find a dynamic person with definite initiative, euphoric, optimistic and choleric. The deficient suprarenal patient is pictured as inadequate, lacking smooth-muscle tone, with a tendency to ptoses of the smooth-muscle viscera. Of pathognomonic import is the "Sargent line," when it can be made to appear on the chest in the immediate neighborhood of the thyroid gland (a white line obtained by stroking the skin and disappearing under epinephrin injection). Hyperadrenia may closely simulate hyperthyroidism, and it is important to differentiate between these two conditions. A patient with the former disease manifests a high blood pressure, less hyperhidrosis, a high red cell count, absence of gastric hyperacidity and a relative degree of bowel stasis.

The clinical cases of pituitary disease are said to be primarily due to a failure of compensatory effort of other ductless glands, notably the thyroid, suprarenal, gonads and pancreas. Timme's discussion of hyperpituitary and hypopituitary states, dyspituitarism and the radiography of the sella, though it presents nothing strikingly original, is well worth careful study.

The gonads are principally and pathologically disturbed through overactive and subinvoluted thymic changes and early pineal deterioraton.

The author's thesis closes with the presentation of an inheritance chart which traces the posterity of union between a giant and a diabetic. Of twenty descendants, there were two with dyspituitarism, one with hyperpituitarism, one with exophthalmic goiter, two giants and one osteomalacic dwarf.

Timme's comprehensive outline is timely. He is enthusiastic—at times perhaps overenthusiastic—but after all, his enthusiasm is founded on original investigation, the research of other students and on clinical experience. Perhaps he is permitting himself too much latitude in the interpretation of certain sets of facts, but after all, inference and speculation, which is the forerunner of more profound knowledge, cannot be too narrowly restricted. In other words, there is permissible and indeed necessary for the formative stage, a certain amount of what has been called scientific guesswork. The certainty that future studies will result in more or less radical modification of the present point of view is recognized clearly by the author. Too much apology for the existing conception of the rôle of the endocrines in neuropathology is out of place. In the first place there are at least several indisputably established disease conditions which rest on as firm a foundation as do typhoid fever or tuberculosis. They provide a scientific basis for more extended investigation. Furthermore, the internal secretory organs lend themselves to accurate physiologic and biochemical study. Surely the field which is being so industriously tilled will yield a rich diagnostic and therapeutic harvest!

STRECKER, Philadelphia.

THE MENDELIAN LAW AS APPLIED TO THE PSYCHOSES, WITH SPECIAL REFERENCE TO SCHIZOPHRENIA. E. Bleuler, Schweizer Arch. f. Neurol. u. Psychiat. **1**:19-40, 1917.

Rudin has finally brought his investigation of the inheritance of the psychoses according to the Mendelian law to a conclusion. He found that of 701 families, including 4,823 individuals, 15.86 per cent. had schizophrenia, and that there were other types of psychosis in 1.63 per cent.; all told, 17.49 per cent. suffered from some form of psychosis, making allowance for those who were too young and might develop symptoms later, and those above the age of 40, when the development of schizophrenia is no longer expected. He found that 4.48 per cent. of persons with practically normal parents suffered from this illness. According to these figures, it is apparent that schizophrenia cannot be an expression of mendelism.

As the younger siblings developed schizophrenia more often than the older ones, it seemed to Rudin that other factors must be present in the production of the disease. It was difficult to determine from Rudin's work exactly what factors were inherited and what psychic manifestations could be considered as hereditary equivalents. It has, moreover, been noted in schizophrenic families that other types of psychoses may appear in the parents; thus, in thirty-five the type was organic, in sixty-five functional, in ten unknown, and in seven alcoholic. Only about one third of the mentally diseased parents of schizophrenic children had the same type of psychosis; and another third had functional disorders, of which about one-third were manic-depressive. While schizophrenic persons frequently have manic-depressive parents, Rudin was unable to find any instance of manic-depressive descendants of a schizophrenic ancestor.

Bleuler criticizes Rudin in so far as the latter selected schizophrenia and dementia praecox for his studies, believing that the disorders could be diagnosed with the greatest certainty. In Bleuler's opinion dementia praecox is of all psychoses, perhaps, the most difficult to be certain of. At the present time, classification of the various subgroups of schizophrenia present almost insurmountable difficulties. Bleuler is convinced that the conception of schizophrenia includes a number of entirely different types of diseases.

Just exactly what should be inherited in these types would be difficult to define. Often nothing is apparent of the disease until the illness has become well developed. We must be able to recognize what he describes as hereditary schizophrenia in contradistinction to manifest schizophrenia; these are related to each other by complex etiologic factors.

To be of definite value an investigation into the heredity of schizophrenia must take the following points into consideration:

1. The conception of dementia praecox, including the hereditary psychosis as well as the clinically manifest psychosis, must be well defined. It must either be uniform in its clinical appearance, or if polymorphic the various manifestations must be well understood.
2. The causal relations between the hereditary psychosis and its manifestations must be known.
3. The diagnosis of the disease must be certain in practically all cases.
4. Those who are the descendants of a homozygote must be manifest schizophrenics for it must be possible to distinguish the dominant and recessive homozygote from the heterozygote.

5. Conditions must permit that all patients in the investigated family can be examined.
6. Heredity must not be limited to either sex.
7. The mortality of the R R-, R D and D D types must be about the same.
8. There may be no shifting of the dominant.
9. No secondary factor may change the figures in any substantial manner.
10. The disease must not appear except through inheritance.

Of these considerations, one to five are completely unknown at the present time; the remaining points are for the most part not well understood. Bleuler thinks also that it is important to investigate not only the families of schizophrenics, but also those of all other types whether psychotic or normal. The relation to alcoholism, manic-depressive insanity and epilepsy must be well understood. The apparent relationship of schizophrenia and alcoholism suggests a polyhybrid genesis of the disease; indeed, the possibility suggests itself that to the schizophrenic gamete must be added some other factors needed to make the disease manifest, in this way really producing an equivalent hybrid.

Woltman, Rochester, Minn.

ANEMIA IN OLD AGE. H. Curschmann, München. med. Wchnschr. **68:** 172 (Feb. 11) 1921.

According to the author, pernicious anemia is considered a disease of middle age. His own observations have led him to believe that this is not the case, as during the past five years he has observed fifteen cases of pernicious anemia in patients whose age varied from 65 to 78 years.

Curschmann contends that many cases are overlooked, especially in the aged, and the symptoms complained of attributed to carcinoma, senile marasmus or cardiac insufficiency. The disease would be more frequently recognized if aged persons complaining of sore tongue were examined for pernicious anemia.

The symptoms complained of in the aged are practically identical to those occurring in patients of middle life. The spinal cord symptoms, such as changes in reflexes, paresthesias, anesthesias, alexia and other symptoms are encountered just as frequently.

The author discusses the subject of aplastic anemia and the possibility of atrophic changes in the bone marrow or other physiologic senile changes being the cause of the changes noted. Hereditary constitutional degenerative changes may also play a part.

Three of Curschmann's cases came to necropsy, but no mention is made of microscopic examination of the central nervous system.

In conclusion the author states: Pernicious anemia is not uncommon in the aged. Frequently the disease is concealed under the mask of senile marasmus and cardiac insufficiency. One should make a blood study of every case of glossodynia occurring in the aged. It is not unusual, as in the middle aged, to have long remissions following the energetic use of arsenic.

Moersch, Rochester, Minn.

THE PROBLEM OF THE PSYCHOPATHIC CHILD. Henry H. Goddard, Am. J. Insan. **77**:511 (April) 1921.

Goddard attempts to formulate certain distinguishing criteria between mental deficiency and juvenile psychopathy. The more important on the basis of the psychametric measurement (Binet-Stanford) and association test (Kent-Rosanoff) are as follows: (*a*) the psychopathic child, while often showing satisfactory comprehension and reasoning ability, is apt to fail in association, weight discrimination, and memory power while the feebleminded child frequently has a rote retentiveness; (*b*) the responses of the psychopath to the Binet questions reveal a prominence of "scatter"; (*c*) the quality of the psychopath's replies are unusual with much individuality and interpellation; (*d*) the psychopath's associations include more than ten individual or more than forty-five common reactions and at least ten abnormal associations.

The further differential diagnosis rests on behavior indications. The points the author makes are excellent, but cooperative study between the psychologist and clinical psychiatrist is needed to decide this phase of the question. Nothing is said of the borderline case. In practice and particularly in outpatient neuropsychiatric work, one is not infrequently confronted with the problem of differentiating from the content between a mere psychotic episode or trend and a beginning psychosis, or again between primary mental deficiency and secondary ill-defined dementia. The decision is especially difficult in the absence of a reliable anamnesis. The presence of "scattering" in the psychometric measurement has considerable value. It may be that Goddard's additional determinations will be even more helpful.

Strecker, Philadelphia.

EARLY APPEARANCE OF SPINAL CORD SYMPTOMS IN PERNICIOUS ANEMIA (BIERMER). Erich Ballmann, Med. Klin. **17**: 249 (Feb. 27) 1921.

The early occurrence of cord changes in pernicious anemia is only superficially touched on in most textbooks. Naegeli, in his latest edition on "Bluterkrankungen und Blutdiagnostik," calls attention to the early appearance of cord symptoms, but is inclined to believe that the blood study if made earlier would have shown signs of the disease.

Ballmann reports four cases in which the cord symptoms were the first indication of trouble; the blood study was made later and the characteristic blood picture discovered. In all cases, paresthesias, gait disturbance and other symptoms long preceded the anemia, and only after careful study was the disease process recognized.

Ballmann is of the opinion that patients showing cord changes are difficult to treat and do not respond as well as those showing little or no cord changes, but a more profound anemia. The four cases reported by the author came to necropsy, and the diagnosis was substantiated.

The author is not certain in what proportion of cases of pernicious anemia cord symptoms are present, but from his personal observations he is inclined to believe that cord changes are more often early than late manifestations of pernicious anemia.

The author brings up the question of fatigue and malnutrition subsequent to the war in relation to cord changes, but draws no conclusions. He finally suggests a careful study of the blood in all system diseases of the cord in which the diagnosis does not seem clear.

Moersch, Rochester, Minn.

THE COMPENSATORY MECHANISM OF DELUSIONS AND HALLUCINATIONS. S. A. SILK, Am. J. Insan. **77**:523 (April) 1921.

This article contains the report of two cases from St. Elizabeth's Hospital and a preliminary and general discussion of the biologic demands in man. The first case is the more interesting. A man, aged 29, was admitted to the hospital suffering from delusions of persecution and reference, "somewhat excessive and circumstantial in speech, and melodramatic in his actions." As a boy he was called a "sissy." Since the age of 12 he had had depressions about once a year. Although well educated, he had failed in several occupations; he then enlisted in the army, where he ran up against a very masculine company commander. After six months' residence, he was discharged as a case of social recovery without insight. The older descriptive psychiatry might say that his life was ruined by manic-depressive attacks; whenever he made a start in business a depression threw him back; he varied between brightness and dulness. The newer view taken by the author regards him as a sickly, spoiled child with a strong mother attachment, homosexual and so insecure, trying to show himself and others that he is not weak by joining the army and building up ideas of persecution about his commanding officer.

BOND, Philadelphia.

VARIATIONS IN INTENSITY OF AFFECTIVE REACTIONS. T. GRAHAM BROWN, L'Encéphale **16**:201 (April) 1921.

Brown gives an account of some experiments dealing with the effect on respiration of stimulation of the midbrain of decerebrate monkeys. The point which he stimulated lay on the transverse section of the midbrain near the aqueduct and at the level of the superior limit of the anterior quadrigeminate bodies. He shows that with a mild current respiration was slowed. As the current was increased, respiration became more and more rapid, until, with a strong current, there were associated movements of the tail, extremities, trunk and neck. He emphasizes the dependence of the reaction on the variation of the strength of the current. He characterizes the respiratory action as an affective one and offers his result as an argument against the statement of Rivers that an "all or nothing" reaction is one of the characteristics of instinct. He ends by saying, "We therefore are brought to the conclusion that an affective reaction may be of varying intensity and that it is not 'all or nothing.'" This work is another link in the chain of evidence indicating that the visceral nervous system is centrally represented by a third neuron, i. e., one central to both the preganglionic and postganglionic portions of the arc. This communication of Brown's is a continuation of work previously published in the *Journal of Physiology,* 1915 and 1916.

KRAUS, New York.

AN EXPERIMENTAL STUDY OF THE SACRAL SYMPATHETIC TRUNK OF THE CAT, WITH SPECIAL REFERENCE TO THE OCCURRENCE OF INTRINSIC COMMISSURAL NEURONS. SYDNEY E. JOHNSON, J. Comp. Neurol. **33**:85-104 (April 15) 1921.

The experiments here described yield accurate information regarding the structure and connections of the sacral sympathetic trunk of the cat, the conclusions from which are clearly presented in a diagram. The normal and degenerated tissues were stained with osmic acid and Ranson's pyridin-silver technic, thus revealing the relations of both myelinated and unmyelinated fibers. The sacral and coccygeal ganglions possess a rich intercellular plexus which completely disappears following descending degeneration of preganglionic axons in the lower lumbar and sacral trunk. It must, therefore, be formed by the terminations of preganglionic efferent axons which run to the trunk through the lower white rami. No evidence was found suggesting the presence of intrinsic commissural or connecting neurons within the sympathetic ganglions themselves or connecting adjacent ganglions. All nervous elements stained by the methods employed were traced to some other source or otherwise accounted for.

C. J. Herrick, Chicago.

TERTIARY SYPHILITIC PSYCHOSES OTHER THAN PARESIS.

Burton Peter Thom, Am. J. Insan. **78**:503 (April) 1921.

The text of Thom's article is that "there is no psychosis which cannot be caused by syphilis." Manic-depressive insanity, involutional melancholia and paranoia are drawn in direct relationship with syphilis. One may readily concede an indirect connection, but the intermediate steps are still uncertain. More and more in psychiatry and indeed in internal medicine, must we subscribe to the doctrine of multiplicity of causes. Even in such well-established conditions as paresis, alcohol or toxic psychosis, we are evidently dealing with syphilis and an extraneous poison respectively plus an unknown factor which preciptates the psychosis. This unknown factor deserves the closest investigation and analysis.

Strecker, Philadelphia.

NERVE AND PLASMODESMA. H. V. Neal, J. Comp. Neurol. **33**:65-75 (April 15) 1921.

The problems relating to the histogenesis of the nervous elements have by no means been fully solved by the demonstration, now generally accepted, that nerve fibers arise as outgrowths of embryonic nerve cells. By many it is claimed that these axonic outgrowths pass into a preexisting protoplasmic network of some sort, a plasmodesma. In favorable material of dogfish embryos stained by the silver-reduction method, Neal has seen neurofibrillae in the primary connections between the neural tube and the myotome, and from his observations he concludes that there are no indifferent plasmodesmas which are utilized as pathways by the growing neuraxons. The earliest structures to appear connecting the neural tube and peripheral end organs are true nerve fibers, axons of the embryonic nerve cells.

C. J. Herrick, Chicago.

REMARKS ON UNDIAGNOSED CASES, CHICAGO STATE HOSPITAL, 1919.

Charles F. Read, Am. J. Insan. **77**:493 (April) 1921.

The more a hospital staff studies its cases the more it leaves unclassified. Read considers the cases of 146 patients under 60 years of age, and it may well be questioned whether he could not have added many beyond that age. His work emphasizes again that the prognosis is better for the unclassified patients than for all admissions. He concludes that confusion and apprehension in an acute psychosis indicate dementia praecox.

Bond, Philadelphia.

ARE THE TASTE-BUDS OF ELASMOBRANCHES ENDODERMAL IN ORIGIN? Margaret H. Cook and H. V. Neal, J. Comp. Neurol. **33**: 45-63 (April 15) 1921.

The specificity of the embryonic germ layers is again attacked in this contribution, in which the authors present evidence that both taste-buds and pharyngeal scales arise in the dogfish from entoderm. These pharyngeal scales resemble in structure the placoid scales of the skin, which are undoubtedly of ectodermal origin. In some other fishes (catfish, carp) taste-buds essentially similar to those within the mouth and pharynx are found freely distributed in the outer skin of the head and trunk, and these have been shown by Landacre to be of ectodermal origin. These observations tend to show that adult structures of similar type may develop from either ectoderm or entoderm, as occasion may arise.

C. J. Herrick, Chicago.

THE FIRST THORACIC WHITE RAMUS COMMUNICANS IN MAN. S. E. Johnson and M. L. Mason, J. Comp. Neurol. **33**:77-84 (April 15) 1921.

Existing descriptions of the white ramus communicans related with the first thoracic spinal nerve are vague and contradictory. Examination of twelve laboratory bodies and of fresh necropsy material showed in each case one or more white rami arising from the first thoracic nerve and connecting, as a rule, with the stellate ganglion. These rami contain a relatively high percentage of large myelinated fibers, whose distribution offers a further problem of some interest. Variations in the arrangement of these rami are described and figured.

C. J. Herrick, Chicago.

TREATMENT OF MELANCHOLIA BY STRYCHNIN IN VERY HIGH DOSAGE. P. Hartenberg, Progrès méd. **36**:123 (March 19) 1921.

Hartenberg points out that melancholic depression is a disturbance of affectivity and of cenesthesia and believes that strychnin in large dosage, by exerting a powerful influence on the vegetative nervous centers, is beneficial. In a series of six cases, he has obtained a cure in five cases and improvement in one.

Using a 1 per cent. solution of the sulphate of strychnin, he commences by giving the first day 7 drops (0.0035 gm. dosage), three times daily, either by mouth or injection. Then, each following day, he increases each dose by one drop. If at the end of several days dizziness and stiffness of the legs and jaws appear, the dose is held stationary until the reaction disappears. Then he again augments the dose as before. One then comes to the point where the same dose always provokes a reaction. This marks the phase of saturation and the dose cannot be further increased. It requires a month approximately to reach the dose (0.07-0.08 gm.) which marks this saturation.

"The progress of the cure parallels the medication." When a dosage of 0.05 gm. is passed, the patient begins to wake, to talk, to occupy himself and to smile.

The writer considers that it is only because strychnin has always been used in too small dosage that its therapeutic value in psychotic depressions has never been recognized

Davis, New York.

FEEBLEMINDEDNESS IN HEREDITARY NEUROSYPHILIS. O. J. RAEDER, Am. J. Dis. Child. **21**:3 (March) 1921.

This paper deals with the incidence of neurosyphilis and feeblemindedness in children of syphilitic parents, and quotes an incidence of 38 per cent. reported by Jeans, and an estimate of 33 per cent. reported by other authors. The author collected thirty cases, the children ranging in age from 2 to 16, whose parents one or both, were syphilitic, most of them neurosyphilitic and usually paretic. In the spinal fluids from twenty-two of the patients, four were positive to the Wassermann reaction and nineteen had a positive blood reaction. The children born nearest the time of the parental infection gave positive reactions while the youngest in a family gave negative reactions. Of the four patients with a positive spinal fluid, the gold curve fell within the "paretic zone." As to the mental condition of these children, those farthest away from the parental infection approached nearest to normal, but it is observed, that though seronegative, they were somewhat backward mentally. The greatest mental defects were found in the older children. In one family in which the father was paretic and the mother became so later on, there were five children ranging in age as follows: 13, 12, 9, 8 and 4. Their mental ages were respectively: $6\frac{8}{12}$, $8\frac{3}{12}$, $7\frac{4}{12}$, $4\frac{10}{12}$ and $3\frac{8}{12}$. The author holds to the view that the syphilitic toxin must have been the cause of the mental defect as the parents were definitely not feebleminded. In another family of four children, the first, who was born before the parental infection, was healthy, the second had a psychosis with all tests positive for neurosyphilis, the third had a positive blood Wassermann, while the fourth was entirely negative. Other children of the series showed a mental age below the actual age, and among them were truants, backward scholars and one with hydrocephalus. One patient only showed a mental age above the actual age.

PATTEN, Philadelphia.

CEREBRAL SOFTENING DUE TO VENOUS THROMBOSIS IN PUERPERAL INFECTION. R. CREMIEU and P. GAUTHIER, Progrès méd. **36**: 101 (March 5) 1921.

This paper includes the reports of two cases of hemiplegia in the puerperium which developed in persons with postpartem infection and pelvic phlebitis. Hemiplegia developed with great deliberateness, in the first case requiring three days. It was possible to exclude syphilitic, eclampsic and nephritic factors as possible etiology. In neither case was there an endocarditis.

The writers believe that whenever a cerebral softening manifests itself in the course of puerperal infection, when the integrity of the heart permits one to eliminate the hypothesis of an embolus, a venous thrombosis is indicated rather than an arterial thrombosis. This diagnosis is rendered plausible by necropsy examinations which can be found reported in the literature, and is strengthened by the known special affinity of the puerperal streptococcus for the venous system.

The patients in the two cases reported both recovered. No opinion is expressed concerning the probable prognosis in most cases.

DAVIS, New York.

PARALYSIS OF THE LEFT RECURRENT LARYNGEAL NERVE ASSOCIATED WITH MITRAL STENOSIS.

J. Garland and P. D. White, Arch. Int. Med. **26**:343 (Sept.) 1920.

In a review of the literature the authors found sixty-one cases of this condition reported. The theories as to the anatomico-etiologic factors are discussed briefly. It is claimed by some writers that enlargement of the left auricle causes pressure atrophy or neuritis of the recurrent laryngeal nerve. Others state that the pressure must be exerted between the pulmonary artery and the aorta or aortic ligament. One writer states that elongation of the nerve is produced by dilatation of the right ventricle. Mediastinitis and thrombosis in the left auricle are given as causes. The authors agree that the pulmonary artery is an intermediate agent in causing pressure, and that occasionally a chronic mediastinitis or a thrombosis in the left auricle are etiologic factors. Auricular fibrillation—by keeping the auricle balooned up, readily producing thrombi, is an added factor.

Nine cases are reported briefly. Summing up the most frequent findings, it is shown that the roentgen ray revealed that three patients had an enlargement of the left auricle. Two patients had enlarged right pupils. Auricular hypertrophy and right ventricular preponderance were present in three, while ventricular preponderance alone was present in two. Four patients had auricular fibrillation. The electrocardiogram of one patient revealed a cardiac displacement from an extracardiac cause. There was possible adherent pericarditis in addition to the mitral lesions in three of the patients.

Hoarseness, cough and dyspnea were present sooner or later in every case, but the duration of hoarseness varied in time before admission from five days to ten months. The authors believe the condition is frequently overlooked.

Patten, Philadelphia.

THE COLLOIDAL GOLD CURVE IN EPIDEMIC ENCEPHALITIS. A PRELIMINARY NOTE.

T. K. Davis and Walter M. Kraus, Am. J. M. Sc. **161**:585 (Jan.) 1921.

A table is given showing the colloidal gold reaction in thirty-four cases of epidemic encephalitis. A study of this table shows that in twenty-five fluids the reaction was definitely negative, giving a zero curve, while in the other nineteen fluids a curve was obtained ranging from 5544332100 to 0000110000. The number of cases showing changes totals 41 per cent. It is noted that the higher curves suggest syphilis, but the serologic studies of the blood and spinal fluid were negative in this respect. The cell counts in the fluids show no parallelism with the colloidal gold curve, ranging from 0 to 134. In some cases three specimens were examined from the same patient, but there were no constant findings. In considering the duration of the disease, the cells seem to be most increased in the third week, but the colloidal gold reaction was less marked at this time than during the seventh to the twelfth weeks. The author concludes that there is no relation between the gold curve and the severity, duration or etiology of the disease. In a fairly large percentage of cases, however, changes of some sort are shown, which indicate a pathologic condition.

Patten, Philadelphia.

THE SPINAL FLUID IN EPIDEMIC ENCEPHALITIS. J. A. Barré and L. Reys, Bull. méd. **35**:366 (April 27 and 30) 1921.

These writers report that in their cases of epidemic encephalitis, chemical and cytologic analysis of the spinal fluid showed hypercytosis in 32 cases, hyperglycosis in 32, hyperalbuminosis in 24 and normal fluid in 19. The maximum cell count was 105. The maximum albumin content 30 mg., and the maximum sugar content 110 mg. per 100 c.c. Increase in sugar alone occurred in 15 cases, of albumin, sugar and cells in 11 cases, of albumin and cells in 9, of cells alone in 8 cases, of cells and sugar in 4 cases, of albumin and sugar in 3 cases, of albumin alone in 1 case.

Thus the meningitis of epidemic encephalitis, which evidences itself clinically infrequently, causes definite modifications of the spinal fluid. The augmentation of the sugar content, however, is the characteristic feature of the meningitic reaction.

Its evolution has no parallelism with oscillations in the clinical course.

The spinal fluid remains abnormal for three, four or even eight months, and for this reason the writers never cease treatment in a case or allow the patient to resume a usual regimen until lumbar puncture proves that the spinal fluid has returned to normal.

Davis, New York.

DISCRETE THERMAL CHANGES IN SPINAL SYPHILIS. Charles A. McKendree, Neurol. Bull. **3**:144 (April) 1921.

McKendree describes a case of spastic paraplegia in a man of 38, probably of syphilitic origin, which came on gradually and was accompanied by urinary disturbance since the onset, three years before. The cord changes were apparently limited to the pyramidal tracts, there being no sensory changes except gross disturbance or loss of appreciation of hot and warm stimuli in both lower extremities. Cold was perceived throughout, and pain sensation was preserved. This unusual dissociation in the appreciation of cold and warm stimuli and pain sensation speaks for a theoretical dissociation of the fibers in the spinal cord, or at least the possibility of their being so discrete that they can be individually damaged. It is rather difficult to explain this dissociation in the spinothalamics which are presumed to carry the pain, heat and cold fibers. The reviewer has seen a similar case in which perception of pain and cold was preserved while that of heat was lost. Further clinical confirmation supported by pathologic findings may lead to more exact knowledge of the sensory pathways in the cord.

Wechsler, New York.

WEICHBRODT'S SUBLIMAT REACTION IN THE CEREBROSPINAL FLUID. Emil Joseph Marx, Monatschr. f. Psychiat. u. Neurol. **48**:227 (Nov.) 1920.

In 1916, Weichbrodt reported his sublimat tests (seven parts of spinal fluid and three parts 1/1000 solution of mercury bichlorid). He asserted that a positive reaction (opalescence, turbidity) meant syphilis. Later he found that certain organic, nonsyphilitic diseases of the brain produced a similar reaction. He found the two reactions different, however—those due to syphilis giving a bluish cast.

Control of these results by other workers has not borne out Weichbrodt's assertion. Marx examined 107 fluids from syphilitic and nonsyphilitic patients, with special reference to the sublimat test. He found it of no especial value. It has approximately the same significance as the Nonne test—phase 1.

Selling, Portland, Ore.

(A) VON RECKLINGHAUSEN'S DISEASE IN THE NEGRO; (B) CURVATURE OF THE SPINE IN VON RECKLINGHAUSEN'S DISEASE. R. S. Weiss, Arch. Dermat. & Syph. **3**:44 (Feb.) 1921.

(a) This is a report of two cases of this interesting disease in the negro. Although occurring in the colored race it does not essentially differ from that seen in white people.

(b) Of greater interest is the report of curvature of the spine associated with this disease in all cases observed at the clinic. There was likewise found in all cases a definite abnormality of the mentality, and in women menstrual disturbance was frequent, which leads the author to suggest the possibility of endocrinous defects as an etiologic factor.

Patten, Philadelphia.

DISAPPOINTMENTS OF ENDOCRINOLOGY. editorial, J. A. M. A.

A recent issue of *The Journal*[1] contains a caution lest, in the current enthusiasm for so-called endocrinology, medicine become humiliated by the drift toward a sort of pseudoscience bolstered up with meaningless words and unfounded assumptions. It is time for the medical profession to face the facts squarely. We shall not deny the great advances that have been made in comparatively few years in the study of thyroid function. Much of the knowledge gained either by scientific research or by clinical observation has "made good" through direct application in practical therapy. Changes in thyroid function may lead to symptoms that are fairly definite, determinable, clinical entities. Therapeutic success attests the value of much that has been accomplished and the verity of a few of the assertions that are taught.

As much cannot be said for the suprarenal structures. G. N. Stewart of the Western Reserve Medical School at Cleveland deserves the thanks of the medical profession for the fearless and critical manner in which he has questioned much of the verbal rubbish that goes under the designation of the endocrinology of the suprarenals. In a trenchant review he[2] has gone even farther in pointing out the invalidity of some of the experimental evidence that has been marshaled to support pet theories. One postulate stands unchallenged, namely, that the loss of the suprarenals is incompatible with life. Hence the conclusion is inevitable that these structures contribute something indispensable to the functioning of the body.

What, then, is "suprarenal insufficiency?" To this question no one has yet given a satisfactory answer. The symptoms attending decline and death after extirpation of the suprarenals are not specific or constant; indeed, they are

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1. The Endocrine Glands.—A Caution, editorial, J. A. M. A. **76**:1500 (May 28) 1921.

2. Stewart, G. N.: Adrenal Insufficiency, Endocrinology **5**:283 (May) 1921.

duplicated by the outcome of various other abnormal states in which no "endocrine" disorder is assumed. The essential manifestations of the loss of the suprarenals remain obscure. Why shall we continue to postulate a condition which cannot be diagnosed even under the most favorable circumstances?

The first answer to any query about the suprarenals is likely to involve the function of epinephrin, the well defined chemical compound that is usually present in the medullary portions and can somehow be discharged into the circulation. The pharmacologic potency of this product cannot be denied. It was natural that attention should be directed promptly to the possibility that epinephrin is the primary active principle, the physiologic hormone of the suprarenals. Is "suprarenal insufficiency" represented, then, by interference with the output of epinephrin? This can scarcely be the case; for animals continue in health after extirpation of one suprarenal and denervation of the other, a procedure after which no detectable amount of epinephrin is liberated. The assertion that epinephrin is at least secreted in physiologic emergencies has been reviewed in *The Journal*[3] with the conclusion of "not proved." The assumption that it serves to maintain the tonus of the blood vessels and the normal blood pressure has likewise proved to be untenable. Injected epinephrin has a definite pharmacologic action on the circulation; so have drugs not even remotely associated with animal tissues. Neither class necessarily represents a normal stimulus because it happens to have demonstrable effects when introduced in the body. No one alleges that the alkaloids, powerful though they are, have a known physiologic function in plants.

Furthermore, although epinephrin is a component of the suprarenal medulla, it is the cortex of the suprarenal structures that appears to be the vitally important portion. If one argues for the "fetish of suprarenal insufficiency," it can scarcely be associated with the hormone epinephrin; for, as Stewart points out, the experimental evidence has tended more and more to show that the cortex is the part of the suprarenal indispensable for life. It is the cortical tissue which alone, or at any rate most conspicuously, undergoes compensatory hyperplasia when a deficiency is created by removal of a considerable part of the suprarenal tissue. The accessory suprarenal tissue, which also undergoes hypertrophy under these circumstances, and which is usually supposed to be responsible for the survival of those animals that do not succumb to the loss of both suprarenals, consists entirely of cortex. Only the cortical tissues can be successfully grafted.

Even in Addison's disease there is no experimental basis for the assumption that the defects of suprarenal function are due to a loss of epinephrin function. How much, then, can be implied in the vague "hypoadrenal" states of clinical literature? There is something stinging, yet deserved in its implied rebuke, in the words of Stewart.[2] "On the whole," he says, "it must be granted that hitherto the attempts made to evoke in animals a well marked syndrome characteristic of adrenal deficiency have been singularly disappointing. The contrast is great when we leave this desert, where the physiologists and experimental pathologists have wandered, striking many rocks but finding few springs, and pass into the exuberant land of clinical endocrinology, flowing with blandest milk and honey almost suspiciously sweet."

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[3] Is Epinephrin Indispensable to Life? editorial, J. A. M. A. **73**:192 (July 19) 1919; The Debated Theories of Suprarenal Function, ibid. **74**:326 (Jan. 31) 1920.

How much longer will our profession continue to merit such criticism? Just so long as our profession continues to give serious consideration to pseudo-scientific rubbish promulgated by the exploiters of organic extracts.

Book Reviews

THE LOGIC OF THE UNCONSCIOUS MIND. By M. K. Bradby. Oxford Medical Publications, London: Oxford University Press, 1920. Pp. 316.

In his introduction to this work the author expresses his thought that the science of logic has to be reformed because we have "outgrown the logic of our forefathers just as we have outgrown their chemistry—a fact which leading logicians are quick to recognize." It occurs to the reviewer that this is hardly a correct statement of the situation. Logicians and students of logic, along with the rest of the population, may always be expected to catch the changing spirit of the times. Just now these logicians and students recognize a demand that logic, along with other disciplines, is required to relate itself to daily life rather than to dead issues. The reforming of logic, to which we are called, is only a new adaptation of the science. The author goes on in his introduction to say that the student of logic is called on to give the subject a fresh start "and this he is enabled to do by the discoveries of psycho-analysts concerning the unconscious mind"—in other words, he might have said that a particular branch of science has brought to light within recent years new data which the logician is called on to reckon with.

The volume under review is taken up with a discussion of these new data which comprise a great body of detail, more or less well verified, that have been brought to light by the psycho-analysts and psychologists of the last several years. Altogether, these data comprise the unconscious background of conscious reasoning which the author discusses in Part 1 of his volume under the titles "Instinct," "Intuition," "Dreams and Unconscious Symbolism," "Language," and "Some Logical Implications of Language."

In all this unconscious background of conscious reasoning the author finds the unconscious motives which are the source of fallacy. These motives he discusses in Part 2 in four chapters entitled, respectively, "Fallacies of Observation and of Conduct," "Some Typical Cases of Fallacy Analyzed," "Fallacies Classified" (two chapters). They are our predisposition, prejudices, etc. Finally, in Part 3 he shows how logic, rewritten in the light of the data supplied by an analysis of the unconscious background, is applied to life; and this he discusses in several chapters entitled, respectively, "Popular Misconceptions Concerning Reason, Logic and Education," "The Logic of Compromise, Logic and Social Problems," "Logic and the Phenomena of Spiritualism, Logic, Love and Religion," "The Logic of God and Devil," "Conclusions," "Progress."

It seems to the reviewer that the present volume is not so much a treatise of logic as it is a discussion of a certain aspect of psychology—that aspect of the new psychology that laps over on the data of the psycho-analyst. This new psychology finds in the data referred to a root of human conduct whether that conduct be the formation of language, the writing of a fable, a dream, the composition of a poem, the attitude toward social problems such as education, or what not. The author has discovered no new logic and no new logical method. He has only pointed out some natural facts, hitherto but partially recognized, that logic must deal with and some pitfalls that it must avoid.

DER EXTRAPYRAMIDALE SYMPTOMENKOMPLEX (DAS DYSTONISCHE SYNDROM) UND SEINE BEDEUTUNG IN DER NEUROLOGIE. Von Prof. Dr. G. Stertz, Oberarzt der Psychiatrischen Klinik in München. Price, 18 marks. Pp. 96. Berlin: S. Karger.

This monograph gives an excellent résumé of the various symptom groups due to disease of the extrapyramidal motor system or systems with some interesting and instructive personal cases. After a brief historical introduction, Wilson's disease and pseudosclerosis are described and compared, the conclusion being that they are essentially the same disease. Then paralysis agitans in its different types and variations is considered, and while the author believes that the anatomic basis of this disease has not been determined, he believes that there is such a basis and that the disease is closely related to Wilson's disease and pseudosclerosis.

The author then takes up in turn the "akinetic-hypertonic" syndrome, double athetosis, torsion spasm (dystonia musculorum deformans), the spastic-athetotic syndrome, the choreic syndrome and myoclonus.

Part 2 covers the application of the knowledge gained by a study of these different extrapyramidal motor disorders to neurologic diagnosis. That is, he shows how a hemorrhage, vascular occlusion, tremor, multiple sclerosis or encephalitis, located in the striatum region, may produce a syndrome very much like paralysis agitans, Wilson's disease, etc. Finally, there is a pathophysiologic summing up of the entire situation with the suggestion that this entire group of diseases be classed together under the designation "dystonic syndrome."

While the author does not claim to cover the literature, his bibliographic omissions outside of Germany are notable. For instance, the papers by Ramsay Hunt are not mentioned, there is only one French reference and Wilson is the only English author quoted—and he is quoted rather grudgingly.

FEEBLENESS OF GROWTH AND CONGENITAL DWARFISM. WITH SPECIAL REFERENCE TO DYSOSTOSIS CLEIDO-CRANIALIS. By Dr. Murk Jansen, O.B.E., Lecturer on Orthopedic Surgery, University of Leiden, Holland. Pp. 82. Oxford Medical Publications. London: Henry Frowde and Hodder and Stoughton, 1921.

This is a further elaboration of principles laid down in previous contributions by the author, particularly in his monograph (1912) on achondroplasia. Part 1 deals with "feebleness of growth" and is a defense of the following three theses: "1. Feebleness of growth is proportional to the intensity of the nocivity. 2. Feebleness of growth is proportional to the rapidity of the growth of the individual and its parts (law of the vulnerability of fast-growing cell groups). 3. Feebleness of growth is characterized by enhanced sensibility and enhanced fatigability."

Part 2 is devoted to congenital dwarfism, with a lengthy and profusely illustrated discussion of dysostosis cleido-cranialis and brief chapters on anencephaly, achondroplasia and mongolian idiocy. All of these conditions are considered as caused by smallness of the amnion becoming operative at various periods of fetal life.

The philosophic and thoroughly logical, as well as original, treatment of obscure developmental conditions makes the book most interesting and stimulating. However, the purely mechanical factors appear somewhat overemphasized, while surprisingly little attention is given to endocrine factors, the importance of which, in disorders of growth, is becoming more generally recognized by other workers in this field.

LEITFADEN DER NEUROLOGISCHEN DIAGNOSTIK. Eine Differentialdiagnose aus dem führenden Symptom für praktische Aerzte und Studierende. Von Dr. Kurt Singer, Nervenarzt in Berlin Neurologischer Fachbeirat beim Hauptversorgungsamt. Paper. Price, 45 marks. Pp. 201, with 33 illustrations. Berlin: Urban & Schwarzenberg, 1921.

The conscience of the author of this little compendium seems to have been a bit uneasy as he takes about four pages to justify his product. Without greatly abusing hyperbole, one might call it a childish book for children. On that basis it may be considered quite useful although it contains little not to be found in any good textbook.

There are eighteen chapters, each devoted to a principal group of symptoms of disease or disorder of the nervous system: paralyses, disturbances of sensation, muscular atrophy, disorders of gait, spasms, tremor, disorders of speech, etc. In general, the diagnostic factors are good, but one receives the impression that the author is neither experienced nor careful. Even a cursory reading reveals errors. For instance, the statement is made (page 17) that a cord lesion below the cervical enlargement, where the nerves for the arms have already been given off but not those for legs, causes a flaccid paralysis of the arms and spastic paralysis of the legs. Facial spasm is (page 80) inextricably mixed with facial tic, and at one place is called impulsive tic, whereas it is neither impulsive nor a tic. The diagnostic criteria of facial neuralgia are pitifully inadequate, the author depending almost entirely on the Valleix points which, as a matter of fact, are generally absent and never of value.

In short, this little book may be helpful to the student and general practitioner (for whom so many sins are committed) but not so helpful as a bigger and better book.

HOW THE MIND CURES A Consideration of the Relationship Between Your Outside and Your Inside Individualities and the Influence They Exercise on Each Other for Your Physical and Mental Welfare. By George F. Butler, A.M., M.D., Medical Director, North Shore Health Resort, Winnetka, Ill.; formerly Professor of Materia Medica, Therapeutics and Clinical Medicine, College of Physicians and Surgeons and Chicago College of Medicine and Surgery. Price, $2.50. Pp. 286. New York: Alfred A. Knapp, 1921.

In the publisher's prospectus it is claimed that "this book tells how the mind cures and in so simple and interesting a manner that anyone who reads it will find that which will teach him how to be and keep well, filling him with delight and giving him a sense of increased power and poise." This is a large order, but it must be admitted that the author has attacked his subject in a sane and conscientious manner and in entertaining form, as a dialogue between the physician and the seeker for all of these benefits. The book gives sensible answers to a great many questions constantly asked by neurotic patients of the educated class and may safely be placed in their hands.

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CALCIFICATION OF THE CEREBRAL VESSELS WITH A CLINICAL PICTURE SIMULATING BRAIN TUMOR*

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INTRODUCTION

In this paper we offer a somewhat detailed clinical and histologic report of a case which is interesting in three respects: First, a diagnosis of brain tumor was clinically justifiable and several operations were performed in the hope of removing the supposed tumor. In other words, it was an example of so-called pseudotumor cerebri. Second, epileptiform attacks of petit mal and grand mal types were the principal symptoms, so the case shows what kind of changes occasionally may cause such attacks. Third, and chiefly, the case is of interest on account of the most unusual pathologic condition presented; namely, a large indurated area in the interior of the left hemisphere in which the capillaries were infiltrated with a hyaline, or colloid material and also calcified to such an extent that decalcification was necessary for the preparation of suitable microscopic specimens.

While calcification of large sclerotic vessels is extremely common as a general process, and while calcification of a single vessel, such as the renal, coronary, or femoral artery is not rare, there are few reports of a condition at all similar to that presented in our case. Mallory[1] relates the case of a woman, aged 45 years, with a clinical diagnosis of chronic nephritis, in which there was colloid and calcareous infiltration of the cerebral vessels, the lime salts being found only in the colloid material. Hansemann[2] reports the case of a man, 28 years old, who had suffered from a speech disturbance, and toward the end also from convulsions, dizziness, headache and vomiting. Colloid degeneration and calcification were present in the vessels of the white matter of both hemispheres, while the large cerebral vessels and all vessels outside the brain were normal. Arnold Pick's[3] patient was still younger, 24 years old, and had suffered from epilepsy from childhood. Later, stupor and signs of tetany were present and brain tumor was suspected. The large vessels were normal, the smaller ones calcified. Similar isolated involvement of larger cerebral vessels is discussed by Marburg.[4]

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*From the pathology laboratories of the Illinois State Psychopathic Institute and Cook County Hospital.

* Read before the Section on Nervous and Mental Diseases at the Seventy-Second Annual Session of the American Medical Association, Boston, June, 1921.

[1] Mallory, F. B.: A Contribution to the Study of Calcareous Concretions in the Brain, J. Path. Bacteriol. **3**:110, 1894.

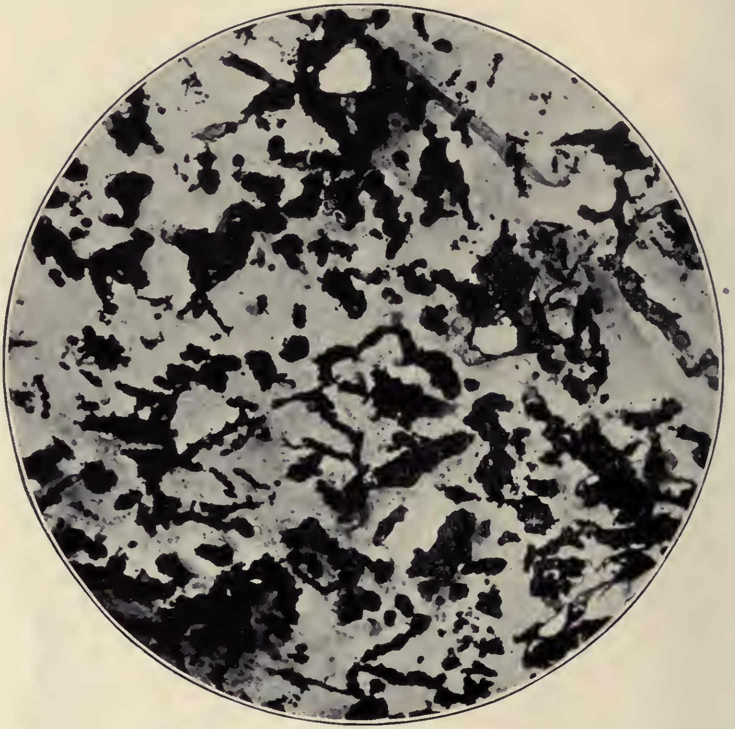

Fig. 1.—A calcified area from the left centrum semiovale in the region of the parietal lobe. Some calcified formations show distinctly a network of petrified capillaries, some are a shapeless mass. Picture under higher power in Figure 2. Thionin stain; X 85.

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[2] Hansemann: Ein casuistischer Beitrag zur Verkalkung der Gehirngefässe, Verhand. der deutschen pathologischen Gesellschaft **2**:399, 1900.

[3] Pick, A.: Calcification of the Finer Cerebral Vessels, with Remarks upon Its Clinical Significance, Am. J. Insan. **61**:417, 1905.

[4] Marburg, Otto.: Zur Pathologie der grossen Hirngefässe, Wiener klin. Wchnschr. **15**:1216, 1902.

while Schmincke[4a] reports calcification of smaller and middle sized vessels of the cortical and subcortical regions in two cases (patients 9 and 18 years old).

Much has been written about "pseudotumor cerebri." One of the best studied cases has recently been reported by Percival Bailey.[5] The symptoms led to exploration for suspected cerebellar tumor, but necropsy proved the case to be one of hemorrhagic meningo-encephalitis with greater involvement of the cerebrum than of the cerebellum. Bailey also gives a concise review of the "pseudotumor" problem and of the more important cases in the literature.

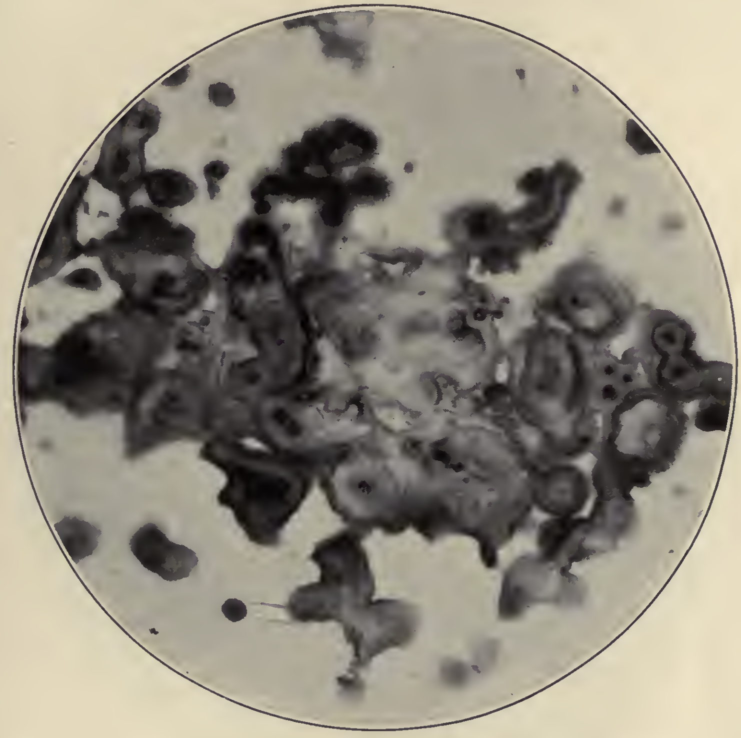

Fig. 2.—A large plaque showing an abundance of calcospherites and capillaries surrounded by a homogenous colloidal substance. One of the areas shown in Figure 1 decalcified with glacial acetic. Van Gieson stain; X 210.

CLINICAL REPORT OF A CASE (DR. BASSOE)

History.—A business man, married, 32 years old, was referred to me by Dr. J. A. Denne, on April 25, 1911. The family history was negative.

As a child he had a "weak back" but later became strong. He had typhoid at the age of 21. He had had frequent epistaxis for years until the beginning of the convulsive seizures. He had had no trauma or venereal disease. He used neither alcohol nor tobacco.

[4a] Schmincke, A.: Encephalitis interstitialis Virchow mit Gliose and Verkalkung, Ztschr. f. die ges. Neurol. u. Psychiat. **60**:290, 1920.

[5] Bailey, Percival: Contribution to the Histopathology of "Pseudotumor Cerebri," Arch. Neurol. & Psychiat. **4**:401 (Oct.) 1920.

The patient began to have light convulsive attacks in March, 1909, which became very frequent, usually from six to eight daily. The first attack was said to have occurred in the night, when he woke up "with a sensation of blood coming out of all the pores on the right side of the face and of the circulation being shut off in the right arm and leg." He could not talk plainly for about half an hour. Such attacks occurred every day. During these he had a vacant, staring expression, and did not answer if spoken to. If he walked during the attack, there was some scuffing of the right foot. The first attack with loss of consciousness occurred in May, 1910, one in February, 1911, and one in April, 1911. Biting of the tongue and frothing at the mouth occurred during these attacks, and once he fell out of bed. The convulsions began in the right side and then became general. Headache and vomiting were not complained of.

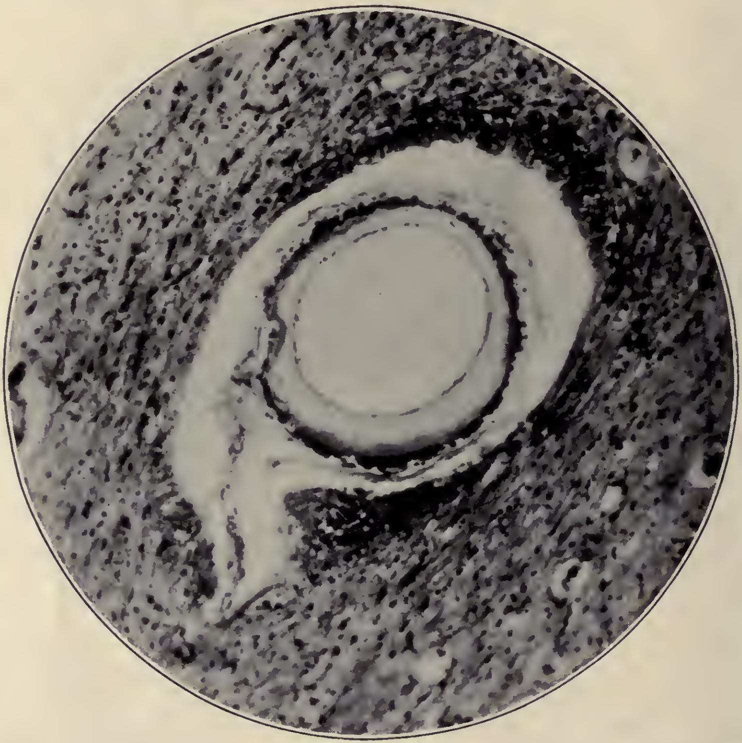

Fig. 3.—A larger vessel of the subcortical area. The Virchow-Robin space is packed with lime salts; the intima and adventitia are well developed, the latter being hyperplastic; the vessel is surrounded by an empty free space, known as the space of His. The latter is bordered by a thickened layer of glia tissue (upper and right parts of the illustration). The rest of the illustration shows numerous glia cells. Bielschowsky's stain; X 170.

Examination (April, 1911).—The pupils and eyegrounds were normal; there was no gross change in the visual fields. The right knee jerk was slightly stronger than the left; the other tendon reflexes were equal and normal. The right abdominal reflex was slightly weaker than the left. The plantar reflexes were normal. Sensation, including stereognostic sense, was normal. The Wassermann test of the blood was negative.

Subsequent History and Treatment.—When seen again in November, 1912, he stated that objects sometimes would fall out of his hand in light attacks, and the right ear felt as if plugged. Involuntary urination sometimes occurred. The right abdominal reflex was now absent, and the wrist and elbow jerks were stronger on the right side. Dr. Brown Pusey found that the poor vision in the left eye, of which he had always complained, was due to hypermetropic astigmatism. The patient complained of trembling of the right hand and difficulty in writing and shaving. In December, 1912, the right abdominal reflex was again obtained, but was weaker than the left one. The cremasteric reflex was present on both sides. The spinal fluid was clear, under normal pressure, gave a negative Wassermann reaction and there was no increase in cells. Six major attacks were observed during the first seven months of 1913. In the lighter attacks he would walk about, dragging the right leg, and would jerk the right arm and shoulder and throw the head to the right.

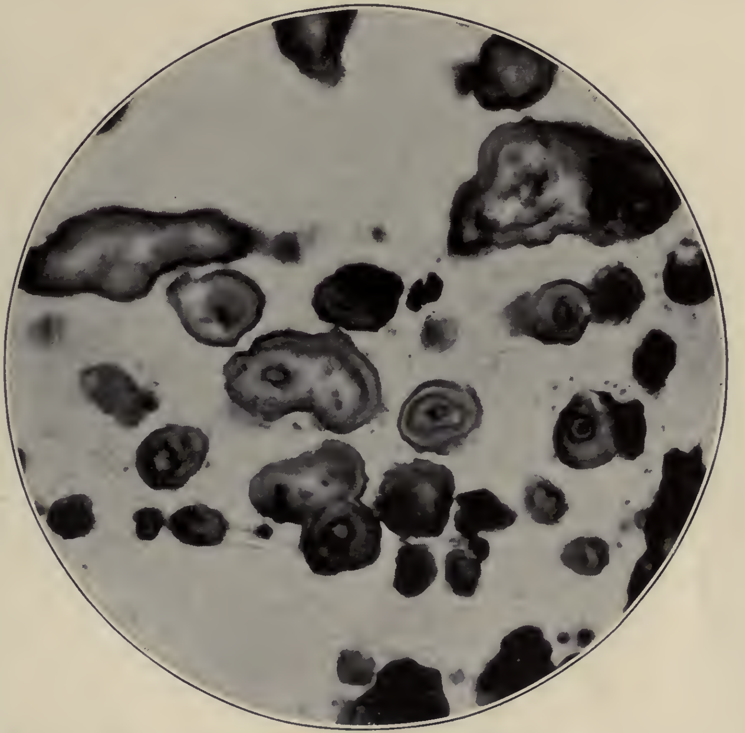

Fig. 4.—A group of psammoma bodies or calcospherites. In some the lamellated structure is marked, some are fused forming larger plaques invested with distinct homogeneous membranes. Some plaques show an abundance of small formations which have no definite structure, such as that of bone or other cell bodies. They evidently are remnants of former capillaries which are quite distinct in the center of isolated calcospherites. The fusion of the latter and formation of plaques can be seen in some areas. Hematoxylin-eosin stain; X 220.

The first operation was performed by Dr. Dean Lewis on Aug. 29, 1913. An osteoplastic flap over the left motor area was turned down. The dura was tense, but the surface of the brain appeared normal. On introducing a needle, resistance was felt one and one-half inches from the surface. After the operation, the right side was weak, and he was aphasic for a few weeks. Later, his condition was as before the operation except that he could use his right hand better, particularly in writing. In October, 1913, his wife reported that he had had several severe attacks during sleep, and that each time he had been lying on his left side. During this time marked pulsation and a bruit appeared at the lower end of the craniectomy wound. In 1914, he had had comparatively few severe attacks and was regularly taking sodium bromid with a little Fowler's solution. The latter was probably responsible for the marked pigmentation of the skin and keratosis of the palms which appeared during the year. In February, 1915, he was sent to Dr. Harvey Cushing.[6] The most noteworthy features at that time were the right-sided weakness, distinct hesitation in speech and general pigmentation. Slight hyperemia and a little blurring at the upper pole of the right disk were noted, and the left was

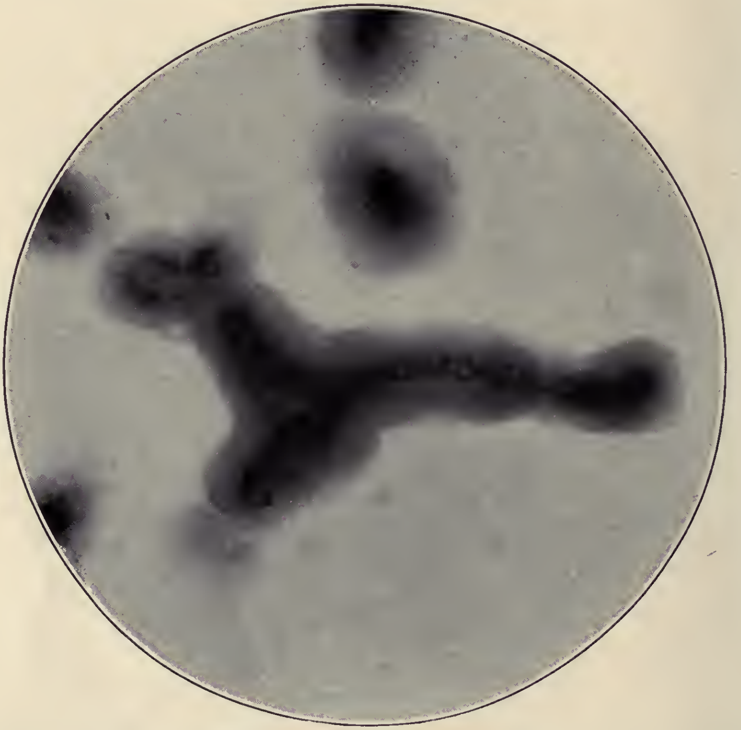

Fig. 5.—Several decalcified (with glacial acetic acid) capillaries surrounded by a homogeneous area of a colloid substance. The larger capillary is branched and shows an abundance of lime droplets over the walls. Toluidin blue; X 425.

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[6] We are greatly indebted to Dr. Cushing for placing his complete record of the case at our disposal.

edematous. The visual fields were normal. All forms of cutaneous sensation were slightly diminished on the right half of the entire body. The patient gave evidence of impairment of memory and of being somewhat euphoric and silly. Spontaneous speech was slow; he frequently hesitated for words and occasionally used the wrong word. The abdominal and plantar reflexes were not elicited, while the knee jerks were active and equal. The right arm was weaker than the left. The grip of the right hand measured 25 kg. and the left, 31 kg. In addition to the slight right hemianesthesia mentioned, there was also diminished stereognostic sense in the right hand. A soft, blowing systolic murmur was heard at the base to the left of the sternum. The blood pressure was: systolic, 178; diastolic, 118.

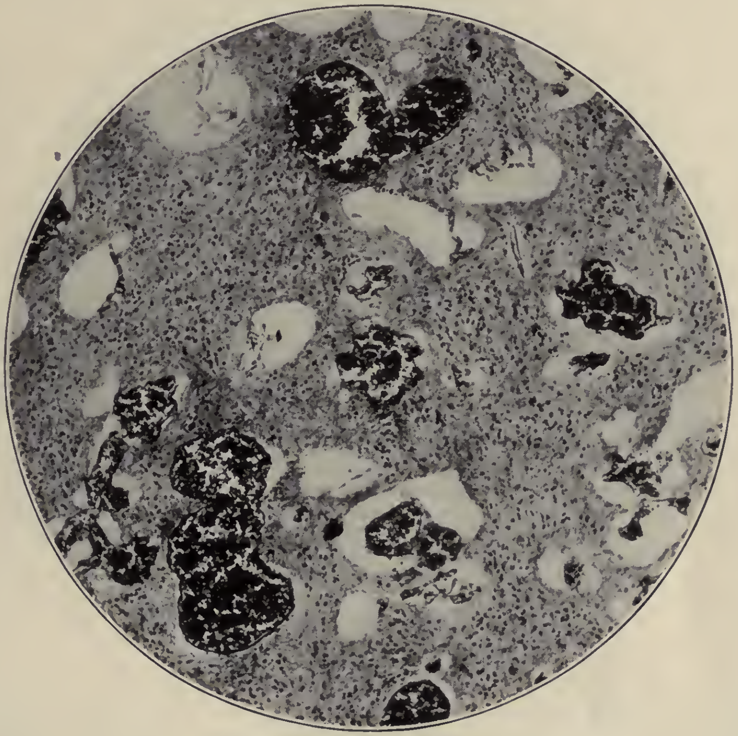

Fig. 6.—An area bordering on the calcified portion of the white substance. A number of hyperemic vessels, mostly veins, fill the large vacuoles, some of which are empty and some of which contain bloodless capillaries. Bielschowsky stain; X 85.

On Feb. 13, 1915, Dr. Cushing performed an operation which consisted of resection of an arteriovenus aneurysm of the temporal vessels, which had developed after the previous operation, and removal of an area of bone posterior to the old wound. The operation was continued on February 19. It was exceedingly difficult to reelevate the old flap and, on account of great loss of blood, the operation had to be abandoned before any subcortical exploration could be made. The patient returned to his home in March, 1915. The attacks continued, and mental deterioration progressed so that he had to give up his work. In January, 1916, he returned to Dr. Cushing. The right-sided weakness and anesthesia were more marked than before.

Ophthalmoscopic examination by Dr. Walker showed that the right disk was paler than the left one and that the margins were slightly faded. The lamina cribrosa was obscured, apparently by a layer of new tissue. There was slight perivascular streaking. The veins had slight residual tortuosity. The conditions suggested a previous inflammation. There was possibly some elevation of the disk.

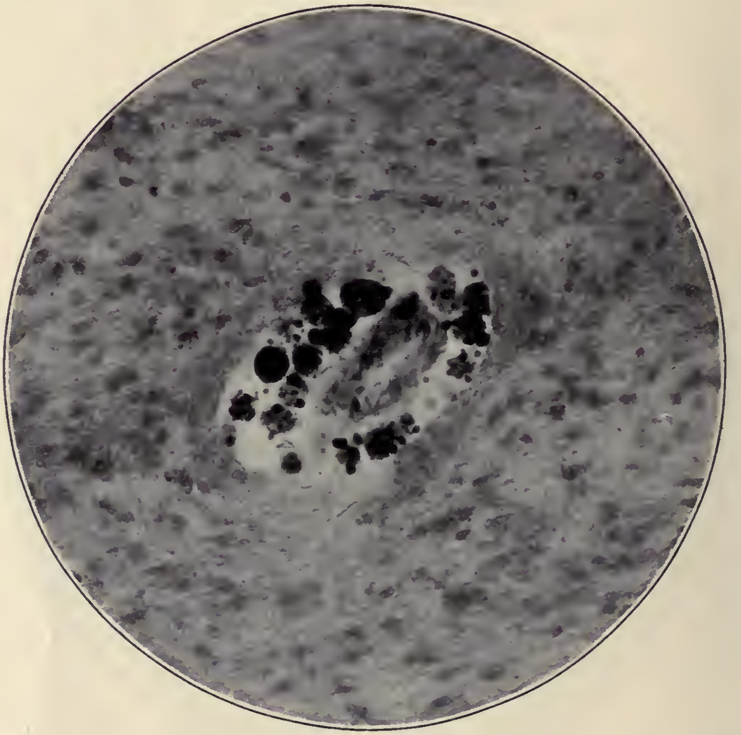

Fig. 7.—A type of small blood vessels scattered over the brain substance. The adventitia is thickened and proliferated, containing in its distended so-called lymph space an abundance of gitter cells packed with large and small drops of fat. Marchi counterstained with Alzheimer-Mann; X 275.

Another operation was performed by Dr. Cushing on Feb. 4, 1916. This time a flap was successfully turned down. The convolutions were flattened and, on palpation, somewhat soft. A needle was inserted in three separate situations and posteriorly it came down to what seemed to be a resistant mass, possibly a tumor. Anteriorly the needle passed without resistance. Although these three punctures were carried down to the depth of the ventricle, no fluid was secured, which led to the inference that the ventricle was compressed and that a tumor probably was present somewhere in the hemisphere. An incision was made posteriorly and carried down to the depth at which resistant tissue had been encountered with the needle. No definite tumor was seen, but the brain tissue was denser and firmer than normal. A small piece of this dense white matter was removed from a depth of about 4 cm. The wound was closed, and the patient made a good operative recovery. Examination of the small piece of tissue removed merely revealed marked gliosis but no evidence of tumor.

There was some subsequent increase in the right-sided weakness, but the attacks for a time were somewhat milder. The mental deterioration increased.

The patient died at his home in May, 1920. Necropsy examination was limited to the brain which was sent to me.

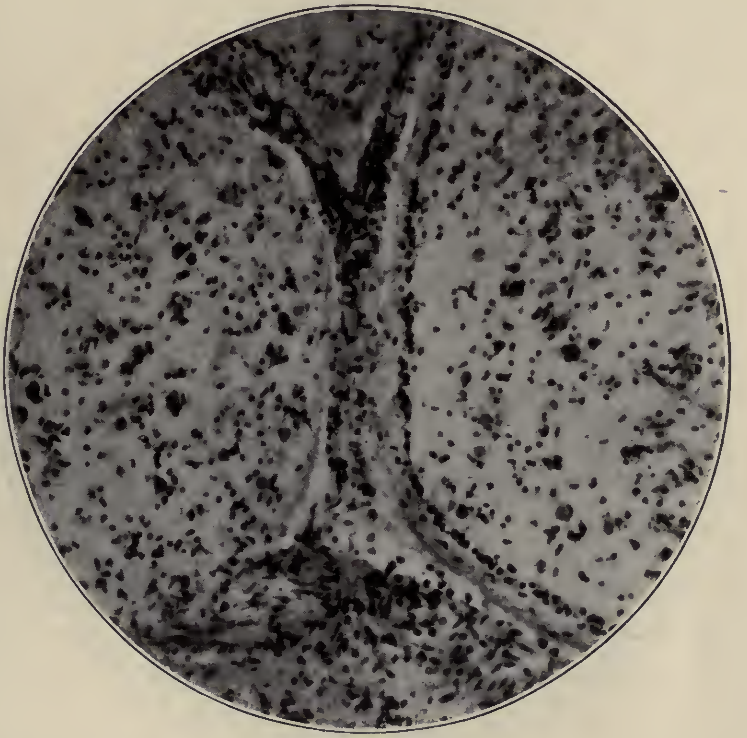

Fig. 8.—Gray substance of corpus striatum; a branched blood vessel with proliferated adventitia surrounded by a dense wall of glia nuclei. The substance shows an abundance of ganglion cells surrounded and invaded by glia cells. Toluidin-blue stain; X 190. (Use hand lens.)

PATHOLOGIC REPORT (DR. HASSIN)

The brain was received in formaldehyde solution. It had been incised when fresh and placed in a small container so that it was in poor condition for macroscopic study. It weighed 1,440 gm. There was a discolored, yellowish area in the pia of the left temporal lobe, and the underlying cortex showed punctate hemorrhages. The convolutions as a rule appeared rather flattened and the sulci shallow. The arteries at the base showed little, if any, thickening. The cerebral ventricles were small. No gross changes were discernible in the right cerebral hemisphere, cerebellum, pons or medulla. On making additional incisions into the left cerebral hemisphere a leathery resistance was encountered and in many places a gritty sound produced. The indurated area measured about 2 by 3 cm. and mainly occupied the basal ganglions and centrum semiovale. It was ill-defined, merging into the surrounding normal substance. The cortical gray matter appeared normal as did the ependyma and choroid plexus.

Microscopic Studies

The microscopic studies covered every portion of the brain. Celloidin, paraffin and frozen sections were stained by numerous methods, such as thionin, hematoxylin-eosin, methyl-blue eosin, Marchi, Bielschowsky and others.

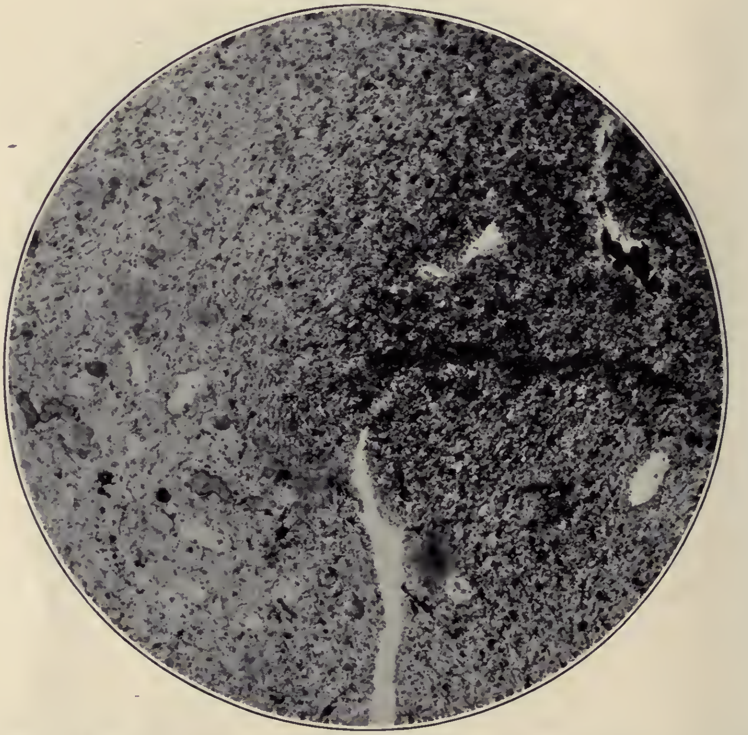

Fig. 9.—White substance (centrum semiovale) showing a black area of fat; the other half shows a few fat droplets (dark spots) and pale calcified bodies invested with a distinct membrane. Herxheimer's scarlet red-hematoxylin stain; X 85.

The white substance, containing the indurated islands, showed numerous homogeneous, shapeless formations which, in some places, occupied the entire field (Fig. 1), totally obliterating the parenchyma. Some sections exhibited more distinct formations in the shape of single round or oval bodies, or of large islands, plaques or conglomerates (Fig. 2). In some places, a distinct network of capillaries could be made out, while in others there were abundant drops and droplets scattered over the parenchyma and often gathered near the blood vessels, constricting their lumen or covering their walls and giving them a marked dotted appearance. Some capillaries were entirely made up of such rows of droplets, while others were only partially covered by them, the droplets evidently becoming confluent and transforming a portion of the capillary into a structureless mass.

The larger vessels (arteries and veins) usually exhibited a normal structure. Some showed within the adventitia the same amorphous substance as that contained in the capillaries. It was confined to the adventitial spaces of Virchow-Robin (Fig. 3), the adventitia being markedly hyperplastic and often showing as a thick homogeneous fibrous ring.

These four types of the amorphous substance—large solid masses forming islands, isolated drops and droplets in the parenchyma, droplets over the capillaries, infiltrations of the adventitial spaces—though quite recognizable in the unstained specimens, were especially distinct when stained. Of the numerous methods used, only that of von Kossa[7] (2 to 5 per cent. of silver nitrate) left them unstained, while Bielschowsky's method (ammonia silver) stained them beautifully. Acids, such as prolonged action of 5 per cent. nitric, hydrochloric or glacial acetic dissolved them with some difficulty, without the formation of bubbles, or rendered them very transparent. Doubtless, we had here to deal with lime deposits. Stained decalcified specimens revealed a great number of

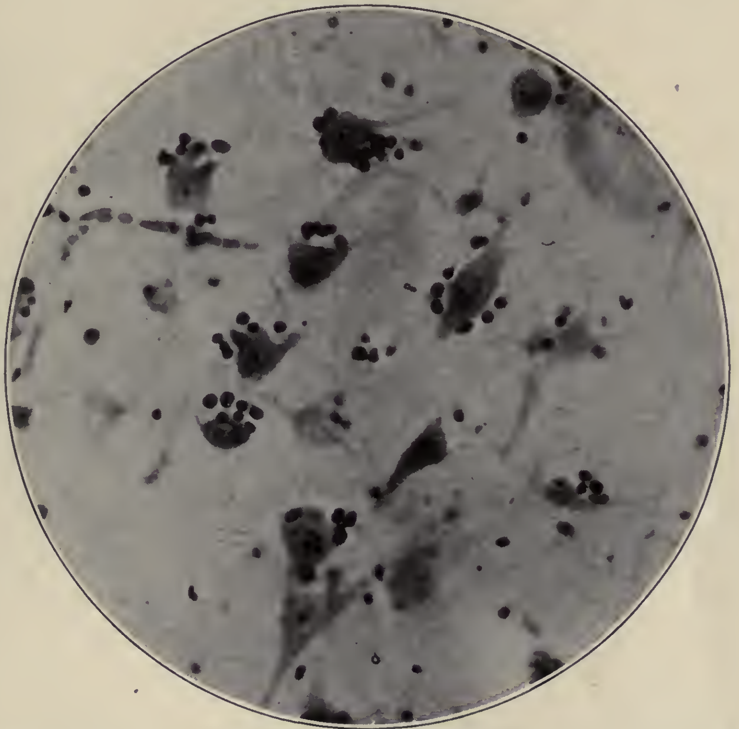

Fig. 10.—Right Ammon's horn. Satellitosis and neuronophagia. Many normal hyperemic capillaries, many are newly formed; absence of perivascular infiltrations. Thionin stain; X 370.

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[7] Von Kossa, J.: Ueber die im Organismus künstlich erzeugbaren Verkalkungen, Ziegler's Beitr. z. Path. Anat. **29**:163, 1901.

round or slightly oval bodies in the form of concentric rings which very much resembled so-called psammoma bodies, corpora arenacea or calcospherites, also known as Harting's bodies (Fig. 4). These bodies were of various sizes, always invested by a thin homogeneous membrane, were scattered singly or formed more or less voluminous islands of irregular shape. The membrane closely followed the contour of the islands and separated them from the surrounding parenchyma. These calcospherites did not give the amyloid reaction, that is to say, they did not stain either with iodin or iodin sulphuric acid, neither did they take the fat stains (scarlet red, osmic acid), but were very well stained with every basic stain and by Bielschowsky's method.

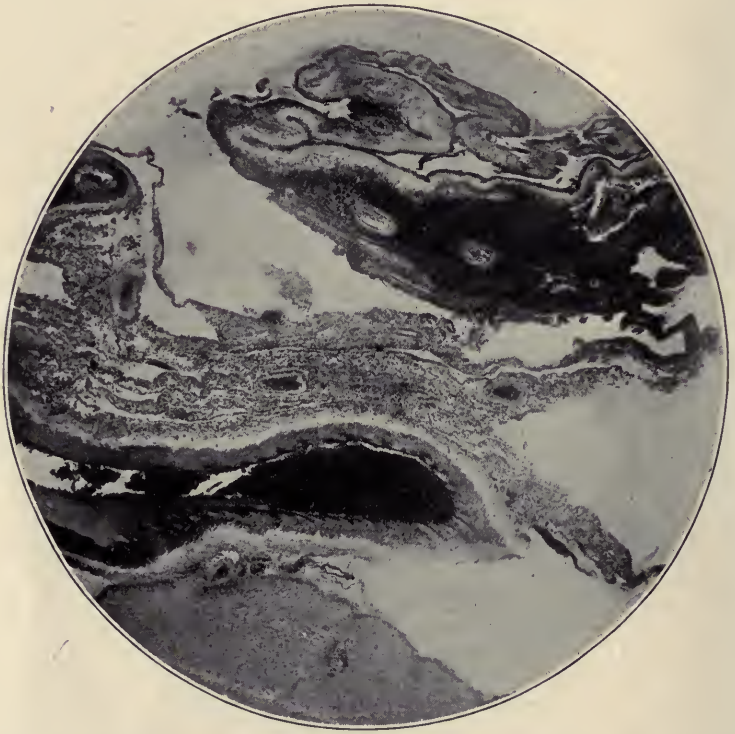

Fig. 11.—Pia-arachnoid with hyperemic veins and meshes infiltrated with blood cells and other elements described in the text. Alzheimer-Mann stain; X 27.

In the capillaries the decalcified specimens showed equally noteworthy features. Photomicrograph 5 shows capillaries possessing a distinct lumen, their walls dotted and surrounded by a broad pale homogeneous substance very much resembling colloid or hyalin. Of the same character is the substance of the islands or conglomerates of the calcospherites in which it occupies a much larger area than the network of the capillaries. The latter are distinctly seen to be the nuclei or centers of the calcified masses.

Aside from vessels surrounded by the amorphous colloidal substance, there was a great number of blood vessels free from any foreign material, but densely hyperemic (Fig. 6) and possessing a thickened wall with well stained nuclei. Especially numerous were such vessels in the areas adjacent to the calcified blood vessels, while other areas even remotely situated from those of calcification showed capillaries infiltrated with gitter cells packed with fat (Fig. 7). Often they were surrounded, muff-like, by a dense ring of delicate glia fibers, or by an enormous mass of glia nuclei, frequently in the form of long rows resembling a wall around the vessel (Fig. 8). Many vessels had thickened adventitia and a greatly narrowed or almost obliterated lumen, while in other places, particularly in the calcified areas, no substance whatever was found, the field appearing vacuolated or areolar.

Areas bordering on those containing the calcified masses, when stained with scarlet red, showed large amounts of lipoid, or fatlike substances, covering the entire field, including the perivascular spaces and the glia, but sparing the nerve fibers (Fig. 9). Such areas appeared altogether red, in sharp contrast to the neighboring blue calcified masses.

Some areas of the white as well as of the gray substance of the large basal ganglions contained numerous foci of fresh and old hemorrhages with a great mass of proliferated adventitial cells, rod cells ("Stäbchenzellen"), plasma cells and pigment granules, mostly contained within macrophages and surrounded by dense walls of glia nuclei. Other areas presented miliary foci of degeneration consisting of sclerosed vessels surrounded by a great mass of gitter cells and macrophages.

Most of the ganglion cells of the basal ganglions in the neighborhood of the calcified areas were normal. However, some showed an abundance of lipoids and neuronophages, the surrounding glia cells being unusually rich in chromatin and often forming clusters (Fig. 8). Among the masses of glia cells were scattered nerve fibers which were thickened, spindle shaped, tumefied, twisted or broken up. The ependyma of the ventricles was proliferated, the subependymal layer consisting of rarefied glia and great masses of glia cells.

Summary of Changes in the White Substance and Basal Ganglions.—The changes found were: calcium deposits around the capillaries of the white substance, calcareous infiltration of the adventitial spaces of the larger vessels, reactive proliferative phenomena in the glia, ependyma and larger vessels, foci of hemorrhages and degeneration in both the gray and white substances and mild parenchymatous changes in the ganglion cells as well as in the nerve fibers.

Cortex.—Comparatively slighter were the changes in the cortex though satellitosis and neuronophagia were quite pronounced. Photomicrograph 10 shows an area from the cornu ammonis densely covered with clusters or islands consisting of ganglion cells invaded or surrounded by glia nuclei.

The white subcortical matter contained an abundance of ganglion cells poorly stained, greatly reduced in size and with shrunken processes. Marchi and scarlet red stains showed fat and pigment in every ganglion cell, as well as in many perivascular spaces. The vessels showed no calcareous deposits, but were greatly hyperemic and often surrounded by walls of glia nuclei, and were decidedly proliferated. In the parietal lobe there was a great number of old capillary hemorrhages such as those noted in the basal ganglions, while in the deeper strata there were congregates of vessels with an abundance of greenish pigment in their adventitia. The minimum changes were present in the frontal lobes, the most pronounced in the cornu ammonis and in the parietal lobe, while the paracentral lobe showed hardly any change. Neither did the cerebellum, pons or corpora quadrigemina exhibit any changes, except some vascular proliferation.

Summary of Cortical Changes.—There were slight parenchymatous changes combined with mesodermic ones, such as hyperemia, vascular proliferation and miliary hemorrhages. As compared with the changes in the basal ganglions, the cortical ones were insignificant.

Pia-Arachnoid, Choroid Plexus and Large Cerebral Vessels (Especially of the Base).—The pia all over the cerebral cortex showed (Fig. 11) distended meshes containing scattered blood cells, numerous foci of hemorrhages and an abundance of macrophages packed with blood pigment and red cells. There were also many mononuclear leukocytes (polyblasts) rich in cytoplasm with more or less intensely stained nuclei and typical plasma cells. The hemorrhagic foci were particularly numerous in the region over the parieto-temporal lobe, less so over the frontal lobe, in which only a few macrophages could be seen loaded with pigment. The vessels of the pia were distended, hyperemic and surrounded with blood pigment but never infiltrated, although the walls appeared thickened, containing oblong nuclei, were well developed and packed with pigment granules. Pigment and some red cells could also be found in the arachnoid membrane which was rich in connective tissue fibers, fibroblasts and clusters of mesothelial cells. Other cells were distinctly reticular in structure much resembling gitter cells. This membrane appeared much thickened, brightly stained, and, like the pia, exhibited marked proliferative phenomena and evidences of old hemorrhages. In addition, both the pia and arachnoid showed abundance of fat granules (Marchi stain).

The choroid plexus showed dense infiltration of its stroma, engorged blood vessels, many of which were calcified, with the lumen entirely obliterated. The epithelial cells covering the blood vessels were of large size and vacuolated, contained an excentric nucleus, black granules and some amorphous substance. A careful study of the basilar and vertebral arteries showed no changes.

GENERAL SUMMARY AND DISCUSSION

The changes outlined were pronounced in the mesodermic tissues (pia-arachnoid, capillaries and larger vessels) and somewhat less marked in the parenchyma (ganglion cell changes and foci of degeneration). Both parenchymatous and mesodermal changes were associated with old foci of hemorrhages and vast deposits of lime (calcifications). The hemorrhages were accidental, the result of three bloody operations. The calcifications were confined to the capillaries of the centrum semiovale and basal ganglions, while the larger vessels only occasionally showed the presence of calcareous masses which were limited to the adventitial spaces. The condition of both the capillaries and larger vessels was altogether unique and different from that observed in syphilis, endarteritis obliterans, so-called calcium metastases, arteriosclerosis and purely inflammatory states. As noted, the calcareous deposits occupied areas infiltrated with a colloidal or hyaline substance; sometimes they occurred in the form of droplets on the surface of the capillary walls, or totally enveloped and destroyed them. In far advanced stages, they formed calcospherites containing broken-up strands and fibers of connective tissue which probably were the sole remnants of former capillaries. As the adventitial spaces of normal.

as well as of calcified, capillaries are in direct communication with those of the larger vessels, it is to be expected that a certain amount of the calcareous substances would be transported to the latter and deposited in their adventitial spaces. The presence of lime salts in the latter would provoke hyperplasia of the adventitia, as well as proliferation of its nuclei, and ultimately lead to fibrous thickening of the vessel wall.

The presence of lime salts in the walls of larger vessels of the brain substance is thus a secondary process which is also true of the capillaries. Being in intimate anatomic association with the tissues, the capillaries are the first to suffer if pathologic phenomena take place in the tissue spaces, tissue fluids or the tissues themselves. If these formations happen to undergo chemical or other changes, such as deposits of colloidal substance, the latter will accumulate around the capillaries, will not be drained by their adventitial spaces, will become stagnant, anchored as it were within the tissues and like any other useless or dead material will ultimately become infiltrated with lime salts. We, therefore, must assume that the tissues harboring the capillaries, as well as the tissue spaces, are the primary sites of the calcareous deposits, and that the calcification is preceded by a stage of some degeneration (colloidal or hyaline). In some cases calcification does not follow the degeneration stage, and the entire pathologic condition is then confined to the stage of colloidal degeneration. This has been demonstrated by Alzheimer,[8] who considers the tissue fluids to be the principal places in which the colloidal masses originate.

These masses are, therefore, to be looked on as a preliminary stage of calcification, an opinion long ago expressed by Holschewnikoff[9] and Mallory, though the former thought that they originate, not in the tissue fluids, but in the capillary endothelium which subsequently undergoes calcification. On the other hand, von Kossa's experiments with calcification of renal arteries in animals led him to believe that the tissue fluids are the places in which lime salts are deposited. The foregoing considerations led us to the conclusion that calcareous deposits, such as were found in our case, are not indicative of some primary vascular disorder, as was thought by Pick and others, but are evidence of some metabolic disorder of the tissue fluids themselves.

The damaged capillaries invaded and covered by calcareous masses were, of course, incapable of nourishing the surrounding tissues. This resulted in necrobiosis with the formation of numerous foci of secondary

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8. Alzheimer, A.: Die Colloidentartung des Gehirns, Arch. f. Psychiat. **30**:118, 1898.

9. Holschewnikoff, D.: Ueber hyaline Degeneration der Hirngefässe, Virchows Arch. f. path. Anat. **112**:552, 1888.

nerve degeneration and the accumulation of large masses of lipoid substances. These lipoid masses drained by the adventitial spaces were carried to the cerebral subarachnoid space, in which they could easily be demonstrated with appropriate stains (osmic acid, scarlet red).

The two foregoing pathologic conditions—calcification and fat accumulation—were associated with purely inflammatory phenomena, such as vascular hyperemia, new formation of capillaries, ganglion cell and glia changes, while infiltrative phenomena were in the background. Such a type of brain inflammation, known as productive encephalitis, is characteristic of toxic states caused by organic or inorganic poisons, such as lead and arsenic. It is highly probable that both the encephalitis and the colloidal degeneration, with the subsequent calcification, were due to the same cause—some severe intoxication.

The latter also plays a prominent rôle in causing so-called ameboid degeneration of the glia, as well as of some gliomatous growths. The ameboid glia strikingly resembles in its physical-chemical aspects colloidal masses, the similarity being so striking that it is impossible to tell the difference under the microscope. Whether or not these two substances are identical is hard to prove, but it is rather suggestive that both are a manifestation of some deep catabolic disorder. Such a disorder probably is also the principal cause of calcification in other regions, the arachnoid, for instance, in which the calcium deposits occur not around the capillaries, but, as demonstrated by Cushing and Weed,[10] in the cells themselves, principally in their nuclei. The conditions in the arachnoid, of course, are different form those in richly vascularized brain tissue; yet the calcification, even in the arachnoid, must be a secondary process, following degeneration of the arachnoid cells.

Wells [11] ably took up the purely chemical problems in connection with calcification in general. He came to the conclusion that calcification is rather a "physico-chemical process beginning as a simple adsorption by hyaline substances which have a more or less specific absorption affinity for calcium." It is, of course, impossible to tell why the abnormal and as yet unknown processes ultimately leading to calcification should affect the white substance only, especially that of the large ganglions, just as it is impossible to explain why these regions are the favorable sites for other abnormal processes, such as Wilson's disease and similar syndromes.

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10. Cushing, Harvey, and Weed, Lewis H.: Studies on the Cerebro-Spinal Fluid and its Pathway, No. IX. Calcareous and Osseous Deposits in the Arachnoidea, Johns Hopkins Hosp. Bull. **24**:366, 1915.

11. Wells, H. Gideon: Calcification and Ossification; Harvey lecture, Delivered March 25, 1911, Arch. Int. Med. **7**:721, 1911.

CONCLUSIONS

1. The white substance of the brain, especially that of the basal ganglions, may be the site of a peculiar degenerative process, followed by calcification.

2. This process is confined to the capillary system.

3. The larger vessels show the same deposits within their adventitial spaces which are invaded secondarily from similar spaces of the calcified capillaries.

4. The lime deposits originate in the tissue spaces which primarily show a hyaline or colloidal degeneration, as the result of some metabolic disorder.

5. The degeneration results in defectvie drainage of the tissue fluids which become stagnant and ultimately calcified.

6. The process is associated with widespread and varied changes in the cerebrum and most likely in the pia-arachnoid. The precise condition of the latter could not be ascertained in our case in view of the traumatic hemorrhages.

7. Clinically, a peculiar mental and nervous condition may obtain which suggests brain tumor.

ABSTRACT OF DISCUSSION

Dr. Percival Bailey, Boston: I recently had an opportunity to examine in Dr. Hassin's laboratory, a brain from a patient operated on by Dr. Harvey Cushing. The patient had had a complete syndrome of the left cerebellar hemisphere. He died some time later, and the brain was sent to me for examination. There was found a meningoencephalitis. In every case examined since Nonne's original communication on "pseudo-tumor cerebri," definite pathologic changes have been found. Dr. Bassoe and Dr. Hassin now establish the pathologic changes in another case. The term "pseudo-tumor" should be abandoned, since it has no clinical or pathologic significance.

Dr. Walter Timme, New York: I was particularly interested in the cases Nonne described in his account of pseudo-tumor cerebri. The pathogenetic factor in some of his cases was a lead encephalopathy as shown by Maas in cases of necropsy. One man was a typesetter; a woman was the wife of a man engaged in the lead industry. There was typical choked disk and all the symptoms of tumor cerebri. Dr. Bassoe, will you give us your findings in cases of lead poisoning?

Dr. Peter Bassoe, Chicago: I believe many of these cases of so-called pseudo-tumor really represent vascular lesions. I recall another patient sent by me to Dr. Cushing for operation in whom only an area of thrombotic softening was found although there was no reason to suspect vascular disease. Another case was that of a man I saw eleven years ago, who presented the symptoms of general increase in cranial pressure including choked disks but no focal symptoms. There was apparent recovery after a decompression and from that time on there was no suggestion of brain lesion, but the patient later developed symptoms of angioneurotic edema. The question arises: May this condition cause symptoms of brain tumor? In regard to Dr. Timme's question about lead encephalopathy, Dr. Hassin made a report at the meeting of the Research Association in Nervous Diseases and spoke on the pathology, which is characterized by proliferation of the small vessels. If we assume that this process is localized, it is easy to understand how a syndrome similar to that of a tumor may be produced.

THE INTRANEURAL PLEXUS OF FASCICULI AND FIBERS IN THE SCIATIC NERVE*

JOHN CHARNLEY McKINLEY
MINNEAPOLIS

During the past fifteen years various writers have developed the conception of fiber tracts within peripheral nerves analogous to those in the spinal cord and brain. That is, in a given nerve, the fibers that supply a certain muscle or a certain area of the skin have been thought to be situated constantly within a definite region of the trunk of the nerve at a given level. Thus, the anatomist Bardeen,[1] from his observations on the segmental distribution of the peripheral nerves of the lower extremity in the embryo and his dissections of nerve trunks in the adult, comes to the conclusion that, though nerves are made up of anastomosing bundles of fibers, the actual course of the fibers is for the most part a straight one from the plexus to the point where a peripheral branch is given off. Further, he thought that a given segmental level of the cord is represented by a certain area of the cross section of a nerve trunk if the nerve arises from more than one segment of the cord. A few years later, Stoffel,[2] apparently without knowledge of Bardeen's observations, developed a similar idea of functional topography in peripheral nerves, basing his conclusions on gross dissection and a study of cross sections of nerves at different levels. He diverges from Bardeen in that he considers fasciculi simply as continuations of the peripheral branches of nerves and therefore thinks it possible grossly to recognize high up in a nerve trunk the exact fasciculi or groups of fasciculi which make up a given peripheral branch. He figures cross sections of nerve trunks showing the arrangement of the bundles according to the branches which he thinks they represent. With this idea in mind he sets down various rules of operative procedure to be followed in the case of nerve suture, transplantation and the like.

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* A thesis submitted to the faculty of the graduate school of the University of Minnesota, in partial fulfilment of the requirements for the degree of Doctor of Philosophy in Nervous and Mental Diseases, 1921.

1. Bardeen, C. R.: Development and Variation of the Nerves and the Musculature of the Inferior Extremity and of the Neighboring Regions of the Trunk in Man, Am. J. Anat. **6**:259, 1906.

2. Stoffel, A.: Beiträge zu einer rationellen Nervenchirurgie, München. med. Wchnschr. **60**:175 (Jan.) 1913. Neues über das Wesen der Ischias und neue Wege für die operative Behandlung des Leidens, München. med. Wchnschr. **60**:1365 (June) 1913. Stoffel, A. und Vulpius, O.: Orthopädische Operationslehre, Stuttgart, 1913. Stoffel, A.: Ueber die Behandlung verletzter Nerven im Kriege, München. med. Wchnschr. **62**:201 (Feb.) 1915.

General interest in this field was lacking, however, until the great war centered the attention of physicians on nerve anatomy and physiology with the hope that nerve therapeutics would be furthered by such new facts as could be ascertained.

A new method investigating this problem was introduced during the period of the war by Marie.[3] By faradic stimulation of nerves exposed at operation and noting the consequent muscular contraction, this writer [4] reached essentially the same conclusions as Stoffel. Other workers [5] have taken up and confirmed many of Marie's findings. The ideas of Stoffel, Marie and their supporters have received general recognition and are much quoted in important treatises on the diagnosis and treatment of nerve lesions.[6]

But this conception of tract systems within peripheral nerves, especially of fasciculi or groups of fasciculi representing peripheral branches of the nerves, has not gone unchallenged. Sherren [7] states that:

> Certainly a third of the (nerve) trunk, may be divided without producing motor or sensory change or one of transient nature only. I have on several occasions divided one third of the internal popliteal nerve without producing any paralysis, often no sensory change, occasionally a loss of power of appreciating light touches which returned in a few days.
>
> * * *
>
> But if the incision cuts into the nerve just above the point of origin of a branch the signs of complete division of that branch are produced. In certain situations also—for example, in the anterior primary division of the fifth cervical nerve—the nerve fibers are arranged in a well-defined order, and incomplete division of this nerve may entail complete division of those motor fibers which supply the spinati and the deltoid muscles. Again, in the trunk

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3. Marie, P.: Des résultats fournis par l'électrisation directe des troncs nerveux dans la plaie opératoire chez les blessés atteints de traumatismes des nerfs, Bull. de l'Acad. de méd. **73**:173 (Feb.) 1915.

4. Marie, P.; Meige, H., and Gosset, A.: Les localisations motrices dans les nerfs périphériques, Bull. de l'Acad. de méd. **74**:798 (Dec.) 1915.

5. Putti, V.: Sulla topografia fascicolare dei nervi periferici e più specialmente dello sciatico popliteo esterno, Clin. chir. **24**:1021, 1916. Benisty, Mme. A. (Translation by Buzzard, E. F.): Treatment and Repair of Nerve Lesions, University of London Press, London, 1918. Kraus, W. M., and Ingham, S. D.: Electrical Stimulation of Peripheral Nerves Exposed at Operation, J. A. M. A. **74**:586 (Feb. 28) 1920. Peripheral Nerve Topography—Seventy-Seven Observations of Electrical Stimulation of Normal and Diseased Peripheral Nerves, Arch. Neurol. & Psychiat. **4**:259 (Sept.) 1920.

6. Tinel, J. (Translation by Joll, C. A.): Nerve Wounds, William Wood & Co., New York, 1918. U. S. Army Manual of Neuro-Surgery, Government Printing Office, Washington, 1919.

7. Sherren, J.: Injuries of Nerves and Their Treatment, William Wood & Co., New York, 1907.

of the great sciatic nerve, the external and internal popliteal nerves remain separate from their origin in the pelvis, hence incomplete division of the great sciatic may cause complete division of the external or internal popliteal nerve.

Heinemann[8] has made dissections of the human median, ulnar and sciatic nerves, following the methods of Stoffel.[2] He concludes that Stoffel's conception is entirely erroneous and misleading. The figures of his dissections show plainly numerous and irregular anastomoses and divisions of the bundles of these nerve trunks throughout their whole lengths.

Langley and Hashimoto,[9] without reference to Stoffel's or Heinemann's work, have made dissections similar to theirs, using the sciatic trunk, the tibial and common peroneal nerves of man and of some of the domestic animals. They describe close plexuses along the course of these nerves and are unable to trace muscular branches proximally, as entities, for any great distance, though certain cutaneous branches remain separate for relatively long distances (24 cm. in the case of the N. cutaneous surae lateralis).

Dustin[10] has made an extensive study of the median, ulnar and radial nerves. He studied them in a way similar to those of Heinemann, and Langley and Hashimoto. In addition he made cross-sections at centimeter intervals so that he could follow their fascicular topography at different levels more accurately than had been done in the past. Kraus and Ingham[5] are the only writers on the subject who have referred to Dustin's work. They state that "the work is purely anatomical and, though of importance, led to no correlation with the functions of the fasciculi he describes." This is true but misleading, as can be seen from Dustin's article:

> 1. The arrangement of fasciculi varies considerably from nerve to nerve and from individual to individual.
>
> 2. The fascicular topography of the same nerve is continually modified by the exchange of numerous anastomoses between the various fasciculi.
>
> From the above it becomes impossible to conceive of a functional systematization of nerves with fasciculi as its basis anatomically. However, one may admit that in every quadrant of a nerve (anterior, posterior, internal and external) such and such motor or sensory fibers are grouped by preference.

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[8] Heinemann, O.: Ueber Schussverletzungen der peripheren Nerven. Nebst anatomischen Untersuchungen über den inneren Bau der grossen Nervenstämme, Arch. f. klin. Chir. **108**:107 (Aug.) 1916.

[9] Langley, J. N., and Hashimoto, M.: On the Suture of Separate Nerve Bundles in a Nerve Trunk and on Internal Nerve Plexuses, J. Physiol. **51**:318 (Sept.) 1917.

[10] Dustin, A. P.: La fasciculation des Nerfs, Ambulance de "l'Ocean" **2**:135 (July) 1918.

One may not, however, subscribe to the exact ideas of Stoffel nor to the conclusions of P. Marie, Gosset and Meige on the existence of constant, distinct fasciculi definitely situated in the nerve trunks.

Results obtained by localized electric simulation are of value only for a given nerve at a given level.[11]

FASCICULI IN THE HUMAN SCIATIC

The right sciatic nerve of an adult human being was dissected and drawn natural size on a piece of plotting paper. Fig. 1 represents this plotting reduced to one-quarter its natural size. The nerve was cut into blocks, 1 cm. in length; these were numbered serially and oriented spatially by stringing them on a thread which passed posteroanteriorly through the proximal end of each. They were imbedded in paraffin and cut serially, still keeping them oriented in space. For the most part sections were mounted at intervals of 3 mm., though two blocks were mounted entire in order to trace the actual anastomoses and divisions of the fasciculi within a limited extent of the nerve (Fig. 6). The sections were stained with hematoxylin and orange G.

The numbers in Figure 1 refer to sections shown in Figures 2 to 5. For example, in Figure 1, Numbers 10, 10′, 10″ indicate the levels of the sections diagrammed in Figure 2, number 10, Figure 4, number 10′ and Figure 5, number 10″, respectively. Figure 6 is drawn from sections taken from above the level at 10 to below that at 11 in Figure 1. Figures 2 to 5 are projectoscopic drawings. The fasciculi are shown in solid black and the connective tissue which cuts the fasciculi into groups is shown by lines.

Examination of Figures 2 and 3, which represent sections at centimeter intervals from the sacral plexus to the bifurcation of the sciatic, shows that no two sections have a similar fascicular topography. There is variation in the number, position and size of the bundles from section to section. Furthermore, the grouping of the fasciculi is variable from section to section in practically every case. The number of fasciculi is variable in the different sections, but in general they become larger and less numerous as the knee is approached, owing evidently to more fusion than splitting. If one then traces the sections out into the common peroneal and tibial it is seen that this fusion continues so that in the common peroneal (Fig. 5, section 5″) there is a single bundle except for three small ones which are about to form the N. cutaneous surae lateralis and a small cutaneous ramus to the skin over the capitulum fibulae. In the tibial the reduction in number of the fasciculi is also marked; there are only three in the tibial trunk proper at the level of Figure 4, Section 8′. If the sections are traced still farther, the fasciculi are seen to redivide into a considerable number of smaller bundles, apparently in preparation for the sending off of branches along the more distal course of the nerves.

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[11] Dustin's article was not available to me until my investigations had been completed (March, 1921). It was at once a disappointment and a satisfaction to note the close correspondence of our observations.

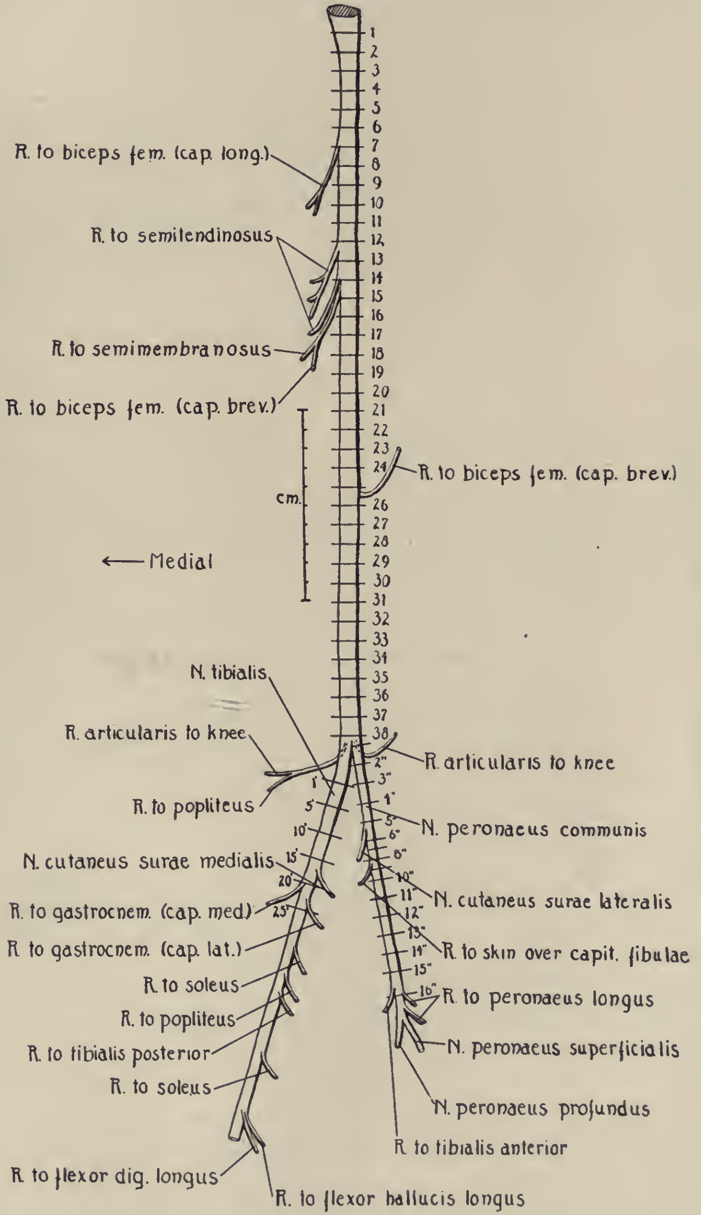

Fig. 1.—Diagram of the right sciatic nerve of an adult human being. The levels of the sections in Figures 2 to 6 are indicated by corresponding numbers in this figure, one-fourth natural size.

The behavior of the fasciculi as they go to form the branches of the sciatic is of interest. While it is probably true that the strands making up the common peroneal nerve do not fuse with any of the tibial nerve bundles and vice versa (we shall return to this in the next section of the article), it is possible to trace the respective groups only about half way up the sciatic trunk before we become uncertain as to whether there is crossing over of the fibers. For example, in Figure 3 at number 38 the peroneal and tibial groups are widely separated; at number 19 or 20 the separation is questionable; and in Figure 2 it becomes practically impossible without sections at closer intervals to follow the two groups as separate entities.

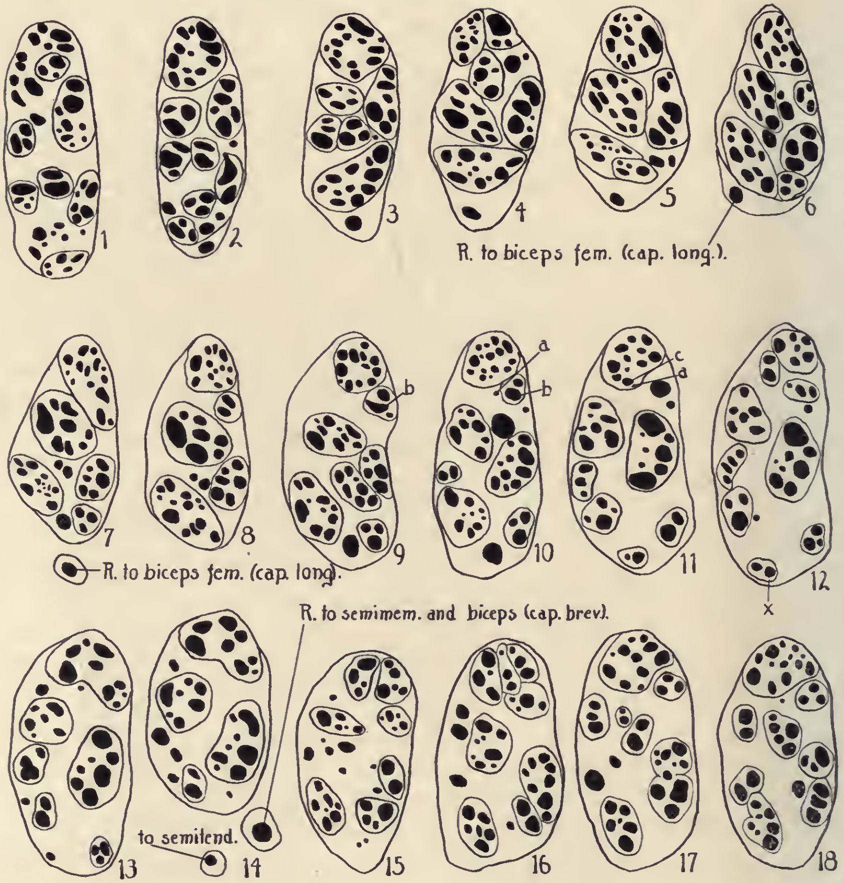
1
2
3
4
5
6
R. to biceps fem. (cap. long.).
7
8
9
10
11
12
a
b
b
c
a
x
R. to biceps fem. (cap long).
R. to semimem. and biceps (cap. brev).
13
14
15
16
17
18
to semitend.

Fig. 2.—Sections at centimeter intervals. Fasciculi are shown in solid black. The connective tissue grouping of the bundles is indicated by the lines. Lateral is to the top of the page, posterior to the right; X 3.

In the case of the fasciculi forming the collateral branches of the sciatic, the tibial and the common peroneal nerves, it will suffice to mention only those branches which are figured as they will serve to indicate the general principles of formation of collateral branches.

In Figure 2, section 7, the branch to the long head of the biceps femoris is seen to be leaving the sciatic. The fasciculus of this branch can be successfully traced to section 2, or a distance of about 6 cm. Between sections 12 and 13 a small branch is given off to the semitendinosus (Fig. 2, section 12 x) which is closely related to another branch to the semitendinosus which is seen leaving the sciatic in section 14. These two branches form fasciculi which fuse when traced proxi-

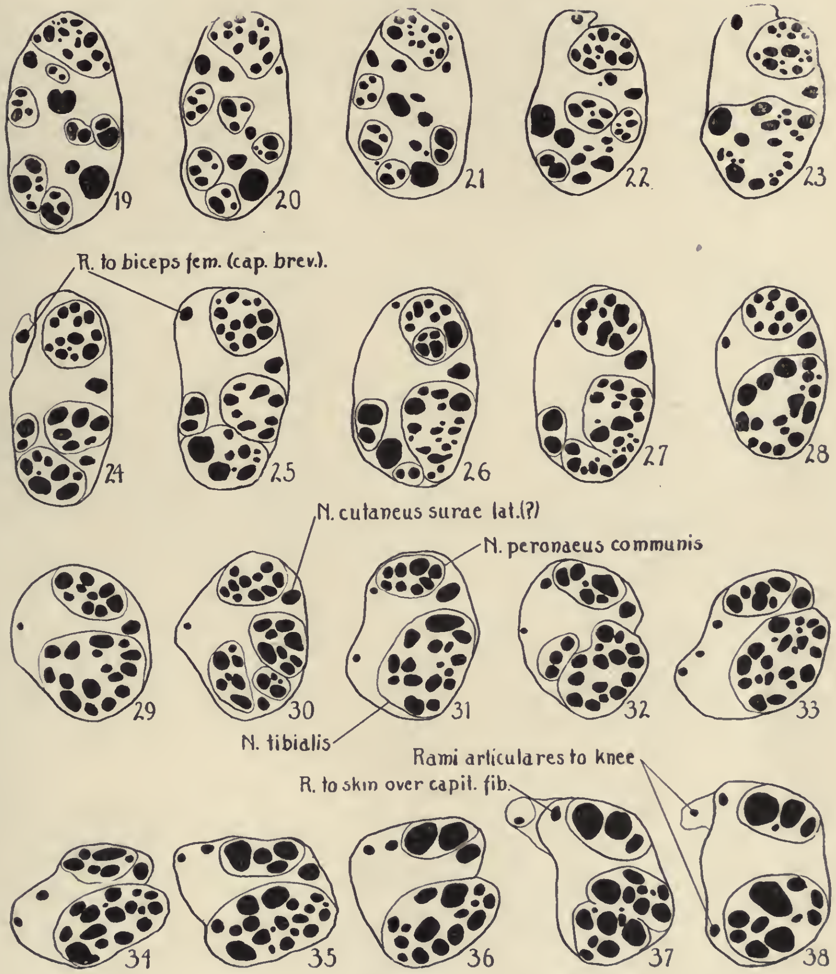

Fig. 3.—Continuation of Figure 2.

mally into one bundle (section 10) which still farther proximally is again broken up (section 8). Thus the fasciculi to the semitendinosus are traceable as anatomically separate pathways for not over 5 cm. along the nerve trunk from the point of departure of the branch. The other collateral branch leaving the sciatic trunk in section 14 runs to the semimembranosus and the short head of the biceps. The fasciculi to this branch can be traced proximally no farther than section 9 (5 cm.). Just distal to section 25 a branch is given off to the short head of the biceps, the fasciculus of which is traceable into section 22 (about 4 cm.). It is conceivable that this bundle could be traced above this point if we had serial sections, but certainly for not more than 4 or 5 cm. additional. Immediately below section 38 two small rami articulares to the knee joint leave the sciatic trunk, one on the tibial and one on the common peroneal side of the nerve. The one on the tibial side is almost immediately lost among the other tibial bundles, but the one on the peroneal side can be traced as high as section 26 (12 + cm.).

Three branches from the tibial trunk are shown in Figure 4. The sections are approximately 3 mm. apart. The N. cutaneous surae medialis is given off immediately below section 17′ (not shown in sections 16′ and 17′ in order to save space on the drawing) and can be traced proximally as a single fasciculus to section 1′ (but not to section 38), about 6 cm. above the point of departure from the tibial. Just below the level of section 20′ a branch is given off to the medial head of the gastrocnemius (see section 13′ for its identification and compare with Figure 1) the fasciculus of which can be traced to section 1′ but not to section 38. The extent of this fasciculus as a separate bundle is thus about 7 cm. The branch to the lateral head of the gastrocnemius leaves the tibial trunk at the level of section 22′ (see section 11′ for its identification) and can be traced also to section 1′, but not higher, a distance of about 7.5 cm.

In Figure 5 are shown sections from the common peroneal trunk at the levels indicated in Figure 1. The N. cutaneus surae lateralis leaves the common peroneal just below section 5″ and is traceable with certainty as a single fasciculus into section 3″, though from the figures it is possible that this fasciculus may extend as high as section 22, a distance of about 21 cm. It is evident that these fibers are located within a definite area of the nerve, while it must be admitted that other fibers probably run along with them (Fig. 3, section 30—N. cutaneous surae lateralis?). The branch to the skin over the head of the fibula leaves the common peroneal nerve near the level of section 8″—the fasciculi can be seen in section 7″. This branch is traceable with fair certainty to section 32; a distance of 13 cm. The branch to the tibialis anterior leaves the common peroneal about 0.75 cm. below section 15″ and cannot be identified in section 14″ so that its fasciculus remains separate from the rest of the nerve for a distance of not over 2 cm. Three small rami to the peroneus longus are seen to be leaving the peroneal trunk at and immediately below section 16″. These branches are represented by three small fasciculi in section 16″ which cannot be identified above section 15″, a distance of about 1.5 cm. In this same section (16″) we note that the common peroneal is dividing into its two terminal branches, the superficial and the deep peroneal nerves. The two corresponding groups of fasciculi apparently remain separate from one another up to sections 8″ or 9″, a distance of about 5 cm.

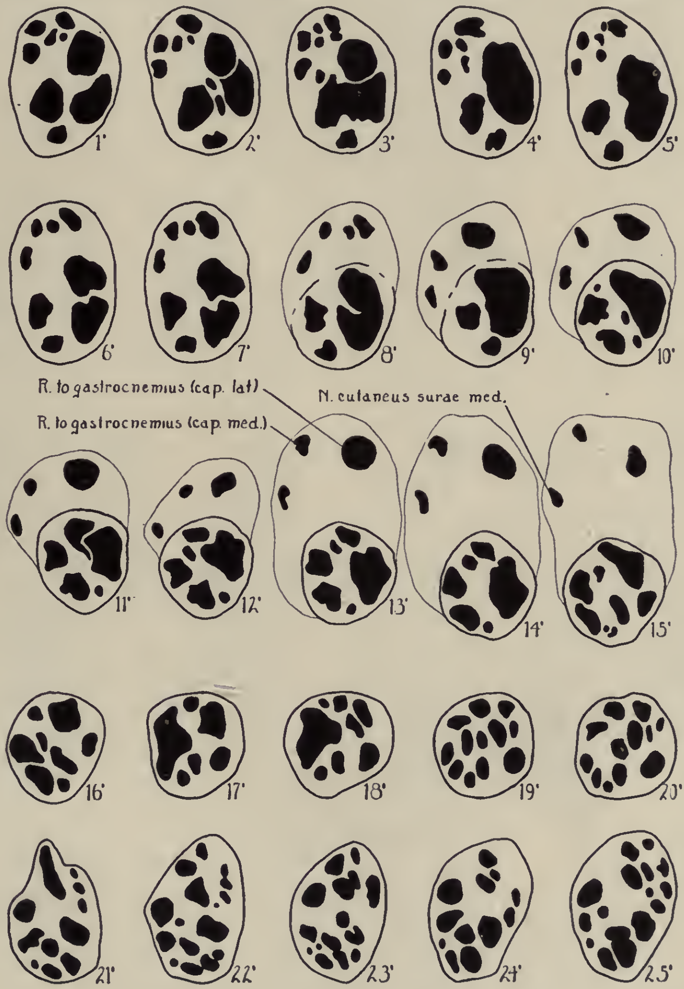

Fig. 4.—Sections at 3 mm. intervals of the right tibial nerve. The numbers of the sections correspond to those in Figure 1. Fasciculi are shown in solid black. Lateral is to the top of the page, posterior to the right; X 4.

It also seemed worth while to determine accurately by serial sections, the exact amount of fusion and division of the fasciculi within a limited extent of the sciatic at a point where the sectional topography does not indicate a rapid shifting of the nerve fibers from one bundle to another. Two centimeter blocks, 10 and 11 (Fig. 1), were chosen for this purpose. Figure 6 is a graphic representation of the results of the study of these two blocks. The sections drawn were taken at sufficient intervals so that a number of anastomoses or divisions of the fasciculi have occurred between any successive two of them. Fasciculi which do not fuse or divide in the course of the nerve between any two of the sections shown are drawn in stipple; fasciculi which do divide or fuse are shown in solid black. Section 1 contains forty-nine fasciculi and section 8 contains forty-one, a reduction of eight bundles within these two centimeters. This reduction would be most simply explained by assuming that eight fusions of the fasciculi had occurred in the region. However, we find that from section 1 to

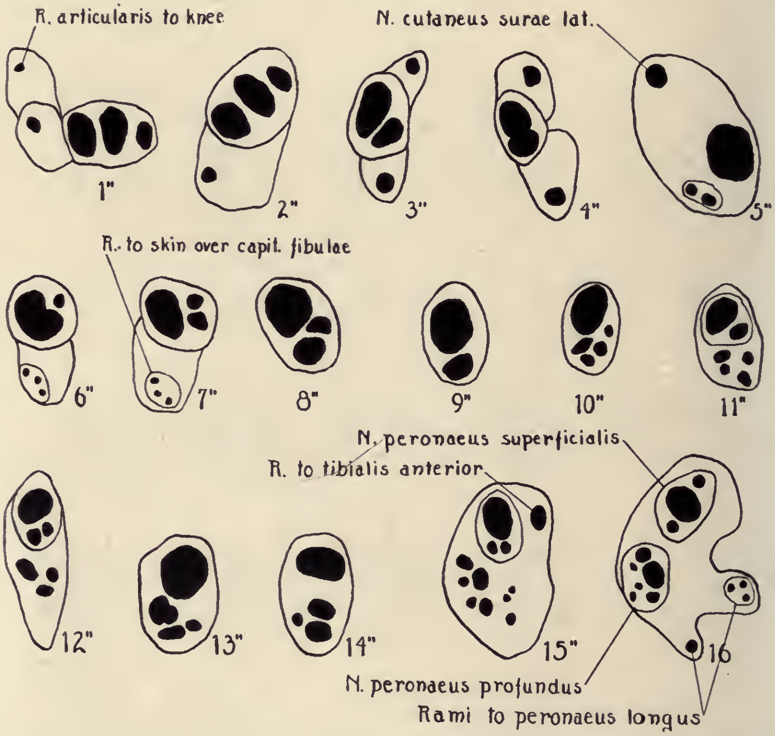

Fig. 5.—Sections of the right common peroneal nerve. The numbers of the sections correspond to those in Figure 1. Fasciculi are shown in solid black. The connective tissue grouping of the fasciculi is indicated by the lines. Lateral is to the top of the page, posterior to the right; X 4.

section 8 there are twenty-three fusions and fifteen divisions, a predominance of eight fusions. Thus, in this limited extent there is considerable passage of fibers from one bundle to another, though on superficial examination it would appear that the fascicular topography did not vary to any marked degree. Another point of interest is indicated in this drawing. The fasciculus marked "a" is an offspring of the one marked "b" (compare with Figure 2). Within these two centimeters "a" moves from the more posterior portion of the nerve, crosses the midline and comes to lie in the more anterior portion, after which it is incorporated in the fasciculus "c". This shows that groups of fibers may quickly cross from one side of the nerve to the other without any marked change in the topographical picture, though as a rule the anastomoses appear to concern adjoining fasciculi.

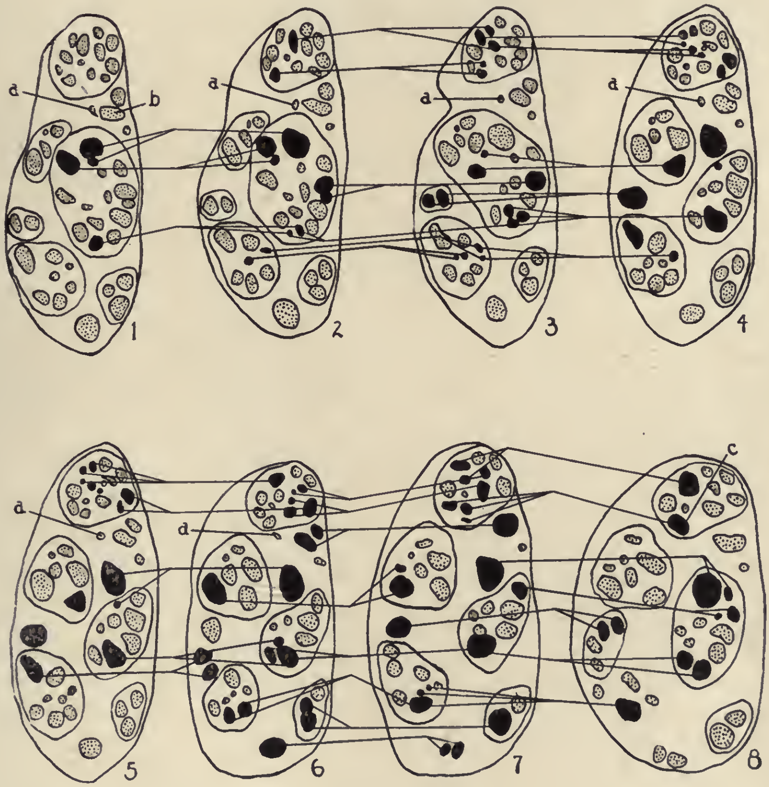

Fig. 6.—Anastomoses and divisions of fasciculi in 2 cm. of the sciatic trunk from above section 10 to below section 11 in Figure 1. Anastomosing or dividing fasciculi are shown in solid black; the others are stippled. Lateral is to the top of the page, posterior to the right; X 4.

The foregoing findings may be summarized as follows:

1. The sciatic is made up of fasciculi which fuse and divide throughout the course of the nerve, forming a vast intraneural plexus.

2. Fusion predominates over division proceeding distally, so that proximally the sciatic is made up of a large number of relatively small fasciculi while in the more proximal portions of its two terminal branches the nerve is composed of relatively few large fasciculi. From about the level of the knee distally, the reverse holds true. The proximal part of the tibial and common peroneal is composed of few large fasciculi which soon split up into a large number of small ones as the more peripheral branches are formed.

3. There is continued crossing of fibers from one fasciculus to another, consequently opportunity for a complete change of functional topography throughout the course of the nerve (e.g., "a" in Fig. 6).

4. Fasciculi in the trunks of the common peroneal, tibial and sciatic nerves may be traced proximally from their respective points of departure for variable distances up the nerve trunks. Aside from the separation of the sciatic trunk into the tibial and common peroneal (which is probably complete), the bundles to the various collateral and terminal branches can be traced a maximum of 21 cm., a minimum of 1 cm. and an average of 6.5 cm. before they are lost in the neighboring fasciculi.

THE COURSE OF DEGENERATED FIBERS IN PARTIALLY DIVIDED NERVES

In order to study the course of the fibers within the sciatic, the following method was undertaken on a number of dogs:

The nerve was bared under aseptic conditions between the ischial tuberosity and the greater trochanter of the femur and was partially divided with a very fine cataract knife so as to cut through various segments of the nerve in the different experiments. The dogs were killed after fourteen days and the sciatic nerves with their branches were stained according to the Marchi method. We could thus study the course of the fibers in a given segment of the nerve just as tracts in the spinal cord have been studied.[12]

Figures 7 and 8 graphically tabulate the results.

Figure 7 shows the degenerations obtained from division of the common peroneal at the level stated. In the figures the degenerated areas of the nerve are shown in stipple, while the normal regions are

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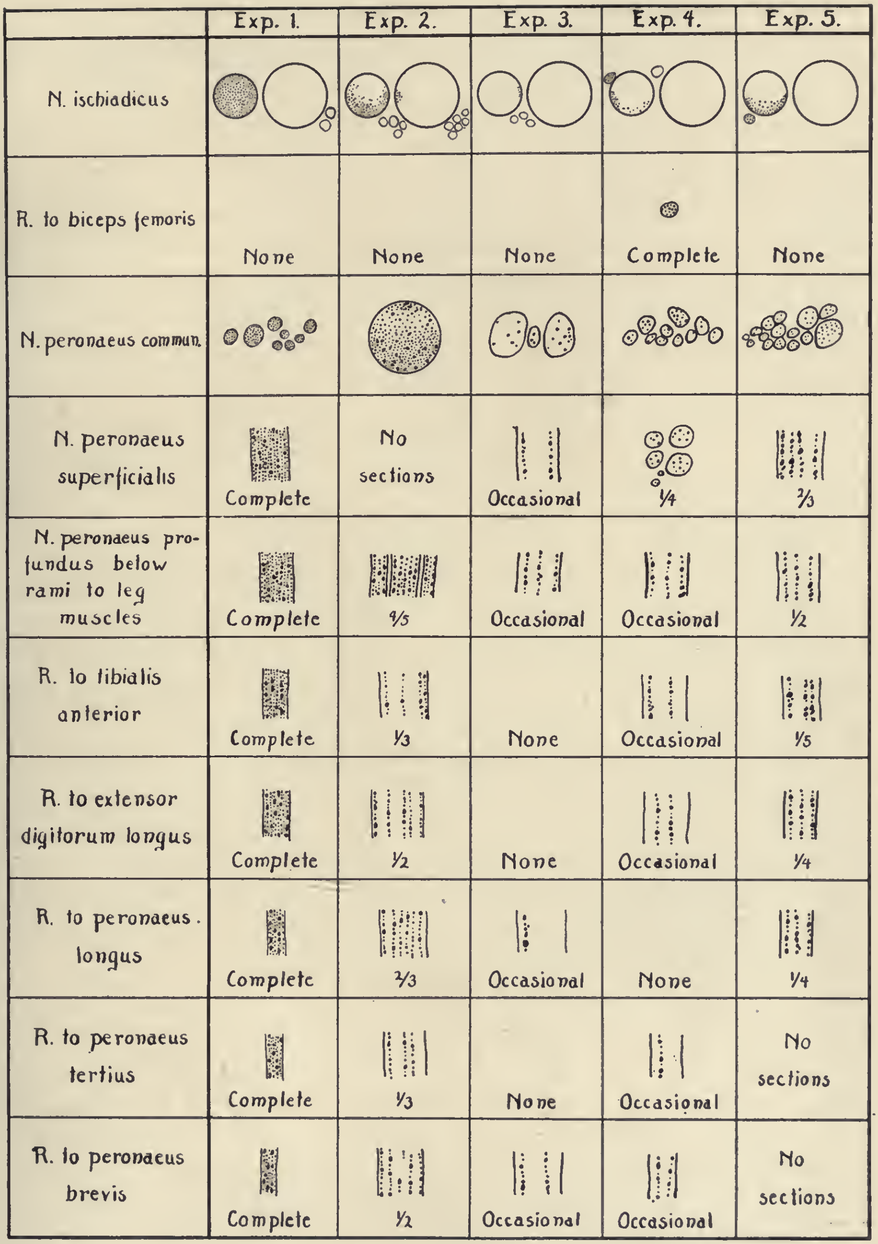
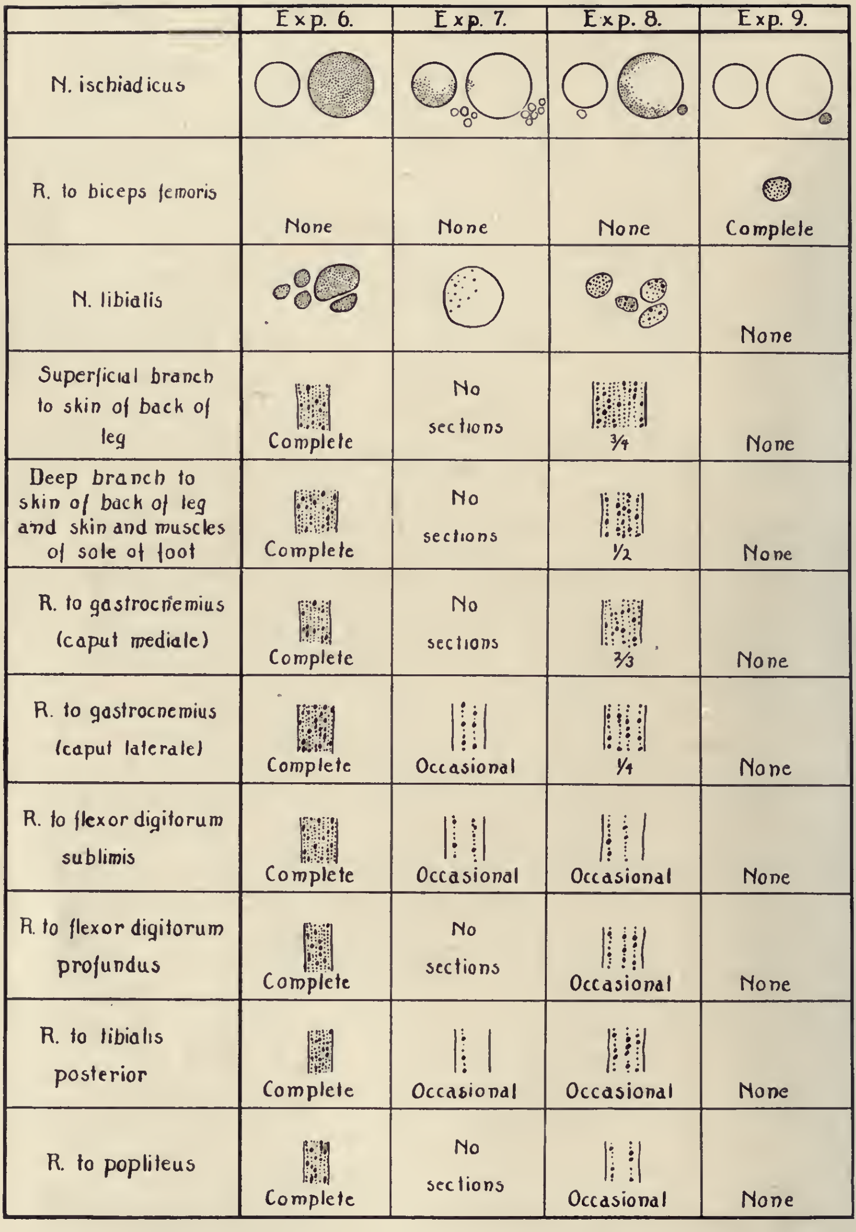
[12] In these experiments free use has been made of Ellenberger, W. u. Baum, H.: Anatomie des Hundes, Verlag von P. Parey, Berlin, 1891. Serial sections in the longitudinal plane were made to determine the presence or absence of degenerated fibers throughout these experiments.



	Exp. 1.	Exp. 2.	Exp. 3.	Exp. 4.	Exp. 5.
N. ischiadicus					
R. to biceps femoris	None	None	None	Complete	None
N. peronaeus commun.					
N. peronaeus superficialis	Complete	No sections	Occasional	¼	⅔
N. peronaeus profundus below rami to leg muscles	Complete	⅘	Occasional	Occasional	½
R. to tibialis anterior	Complete	⅓	None	Occasional	⅕
R. to extensor digitorum longus	Complete	½	None	Occasional	¼
R. to peronaeus longus	Complete	⅔	Occasional	None	¼
R. to peronaeus tertius	Complete	⅓	None	Occasional	No sections
R. to peronaeus brevis	Complete	½	Occasional	Occasional	No sections

Fig. 7.—Marchi degeneration following partial division of the common peroneal nerve. The branches under consideration are indicated in the column at the left. Degenerated fibers are shown in stipple; normal areas are left blank. "N. ischiadicus" indicates a section of the nerve just below the level of the lesion. "N. peronaeus communis" indicates a section of the common peroneal at the level of the knee. Anterior is to the left, lateral is to the bottom of the page for the sciatic and common peroneal sections.



	Exp. 6.	Exp. 7.	Exp. 8.	Exp. 9.
N. ischiadicus				
R. to biceps femoris	None	None	None	Complete
N. tibialis				None
Superficial branch to skin of back of leg	Complete	No sections	¾	None
Deep branch to skin of back of leg and skin and muscles of sole of foot	Complete	No sections	½	None
R. to gastrocnemius (caput mediale)	Complete	No sections	⅔	None
R. to gastrocnemius (caput laterale)	Complete	Occasional	¼	None
R. to flexor digitorum sublimis	Complete	Occasional	Occasional	None
R. to flexor digitorum profundus	Complete	No sections	Occasional	None
R. to tibialis posterior	Complete	Occasional	Occasional	None
R. to popliteus	Complete	No sections	Occasional	None

Fig. 8.—Marchi degeneration following partial section of the tibial nerve. The branches under consideration are indicated in the column at the left. Degenerated fibers are shown in stipple, normal areas are left blank. "N. ischiadicus" indicates a section of the sciatic trunk just below the level of the lesion. "N. tibialis" indicates a section of the tibial trunk at the level of the knee. Anterior is to the left, lateral is to the bottom of the page for the sciatic and tibial nerve sections.

left blank. Thus, in Experiment 1 the whole common peroneal nerve was cut across, and we see a corresponding complete degeneration of all the fibers contained within it. Each of the branches of the common peroneal is likewise totally degenerated. No degeneration was found in the tibial nerve trunk nor in any of its branches.

In Experiment 2 approximately the lateral half of the common peroneal was divided, and we see a corresponding degeneration with the Marchi stain. The heading "N. ischiadicus" indicates a section of the sciatic 1 or 2 cm. below the level of the lesion. At the level of the knee the degenerated fibers are scattered throughout the whole diameter of the peroneal trunk. These diagrams may be checked by the photomicrographs shown in Figures 9 and 10. Figure 9 corresponds to the drawing in Figure 7, Experiment 2, N. ischiadicus; Figure 10 corresponds to Experiment 2, N. peronaeus communis. There is degeneration in all sectioned branches of the common peroneal. About four fifths of the fibers in the N. peronaeus profundus below the point where the rami to the leg muscles are given off are degenerated, about two thirds of those in the branch to the peronaeus longus, about half the fibers to the extensor digitorum longus and the peronaeus brevis, and one third of those to the tibialis anterior and the peronaeus tertius are degenerated. Figure 11 is a photomicrograph of the branch to the

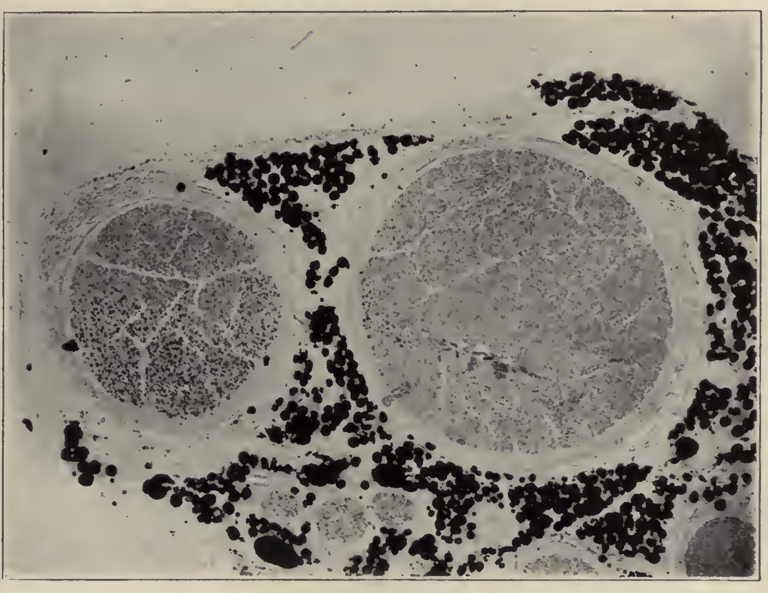


Fig. 9.—Photomicrograph of a Marchi section of the sciatic nerve just below the level of the lesion in Experiment 2. Posterior is to the right, lateral to the bottom of the figure; X 26.

peronaeus longus and may be used for comparison with the drawing of the degeneration of this branch shown in Experiment 2, N. to peronaeus longus (Fig. 7). In this same experiment there is slight degeneration of the anterior part of the tibial nerve which is traced out in Figure 8.

In Experiment 3 (Fig. 7) only a few fibers of the posterior side of the common peroneal were divided (see under N. ischiadicus). No degeneration is demonstrable in the branches to the biceps femoris, the tibialis anterior, the extensor digitorum longus and the peronaeus tertius. In sections of the N. peronaeus communis the degenerated fibers are considerably scattered throughout the cross section of the nerve. Degeneration is demonstrable in about equal amounts in the N. peronaeus superficialis, the N. peronaeus profundus below the rami to the leg muscles, the ramus to the peronaeus longus and to the peronaeus brevis.

The common peroneal in Experiment 4 shows compact degeneration in the anterior part and a few scattered degenerated fibers along the lateral side of the nerve. Also, a small fasciculus of the sciatic accompanying the common peroneal along its anteromedial side is completely degenerated. The branch to the biceps femoris is completely degenerated and is undoubtedly represented by the small fasciculus just mentioned. The N. peronaeus superficialis is about one quarter degenerated; there is no degeneration in the branch to the peronaeus longus and occasional degenerated fibers are found in each of the other rami studied.

In Experiment 5 the lateral third of the cross section of the common peroneal is degenerated, together with a small accompanying fasciculus which is completely degenerated. No completely degenerated branches are seen in this nerve so it must be assumed either that some branch (probably to the biceps femoris) has been lost in the dissection or that this fasciculus has fused and been incorporated again with the common peroneal. From a study of other sections in this series it is most probable that the former is the correct explanation. The N. peronaeus superficialis is about two thirds degenerated, the N. peronaeus profundus below the points of emission of the branches to the leg muscles about one half degenerated, the branches to the extensor digitorum longus and peronaeus longus each about one quarter, and the branch to the tibialis anterior about one fifth degenerated. No sections of the branches to the peronaeus tertius and brevis are available because of difficulty in dissection.

Figure 8 shows results of total and partial divisions of the tibial trunk. The caption "N. ischiadicus" again indicates a section of the sciatic trunk taken just below the level of the lesion. In Experiment 6 the tibial has been divided completely. We find correspondingly complete degeneration of the tibial trunk and all its branches. There is no demonstrable degeneration in the peroneal division nor in any of its branches.

Experiment 7 is from the same nerve that is shown in Experiment 2, but here the tibial degeneration is considered. We find that the small group of degenerated fibers at the anterior margin of the tibial division at the level of the lesion is scattered over at least the anterior half of the tibial trunk when sectioned at the level of the knee. Through some error sections of only three branches of the tibial are available in this experiment. However, all three of these rami show degenerated fibers.

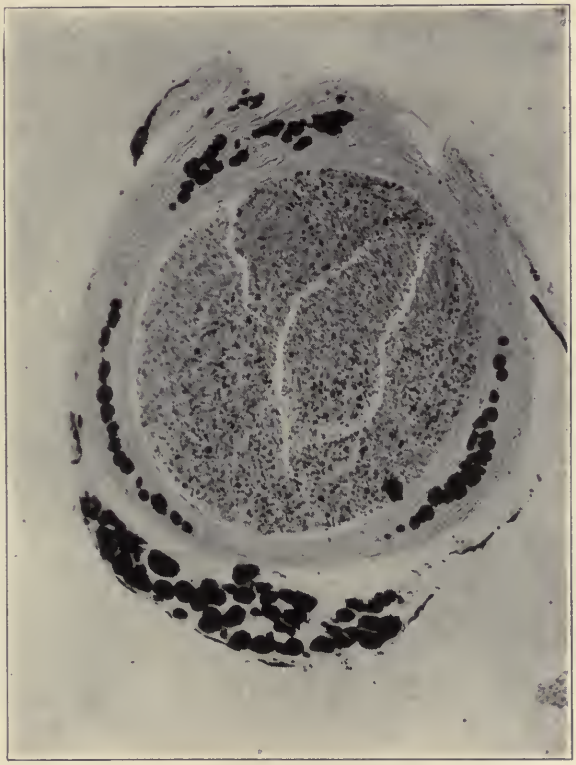


Fig. 10.—Photomicrograph of a Marchi section of the common peroneal nerve at the level of the knee in Experiment 2. Posterior is to the right, lateral to the bottom of the figure; X 46.

In Experiment 8 the tibial trunk was divided along its lateral, anterior and medial margins (Experiment 8, N. ischiadicus). A small fasciculus accompanying the tibial side of the sciatic was also completely divided, and we are unable to trace this fasciculus into its terminal branch. The explanation in this case is the same as that already given for an analogous finding in Experiment 5. We see in this experiment (8) that at the knee level the tibial nerve is partially degenerated throughout its whole cross section. Also there is degeneration in every branch of this tibial nerve though most marked in the superficial branch to the skin of the back of the leg, in the ramus to the gastrocnemius and in the deep branch of the skin of the back of the leg and the skin and muscles of the sole of the foot.

In Experiment 9 only a small fasciculus was divided. The only branch of the sciatic found degenerated was a small one to the biceps femoris.

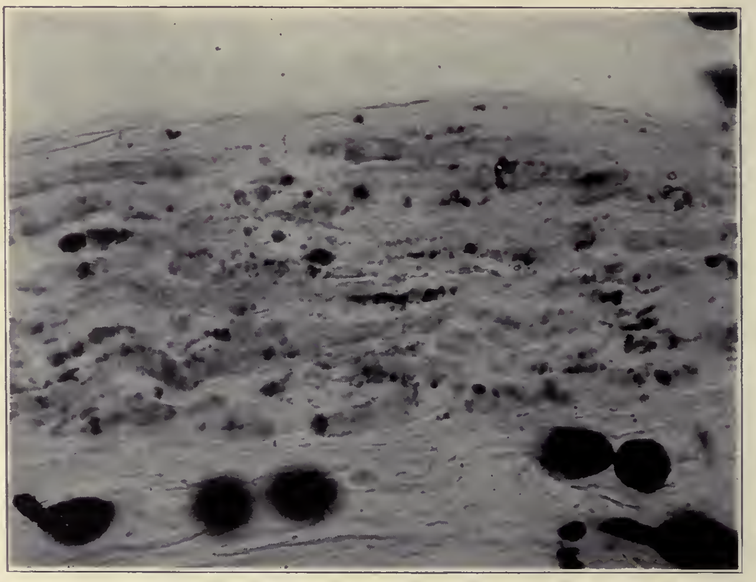


Fig. 11.—Photomicrograph of a longitudinal section of the nerve to the peronaeus longus in Experiment 2. The figure shows the typical appearance of degenerating myelin sheaths in longitudinal section when stained according to Marchi's method. About two-thirds of the fibers are degenerating; X 300.

These experiments show that in the dog the tibial and common peroneal are completely separate from one another at least as high as the level of the ischial tuberosity and the greater trochanter of the femur. Also, partial division of the common peroneal or tibial gives regularly a degeneration which spreads out over most of the cross section of the nerve in its course down the sciatic trunk and is demonstrable in all of the peripheral branches if more than a very small area is divided. The fasciculi to the hamstring muscles are given off for the most part above the level at which these experiments were performed though occasionally a small fasciculus to the biceps femoris is seen coursing along with, but remaining separate from, the sciatic trunk for a short distance.

It is regrettable that the foregoing observations are so few and that they have been made on the dog, in which the tibial and common peroneal divisions consist each of a single bundle. Probably more satisfactory results would have been obtained had the goat been used, whose sciatic morphologically more closely resembles that of man and in which partial division of the nerve would have been simpler because of numerous fasciculi more or less visible to the naked eye (Langley and Hashimoto [9]).

ELECTRIC STIMULATION OF THE SCIATIC TRUNK IN THE DOG

A single experiment was done. In an anesthetized dog, the tendons around the ankle were dissected out and each connected by means of a string to its respective lever playing on a drum of smoked paper. The sciatic was isolated between the ischial tuberosity and the greater trochanter of the femur, from 3 to 4 cm. below the sacral plexus, was cut across and stimulated by means of an ordinary induction coil with small electrodes. The current used was the minimal to produce contraction of the muscles. In Figure 13, No. 1, is shown the tracing obtained on passing a current through the whole cross section of the nerve. All the muscles of the leg contracted.

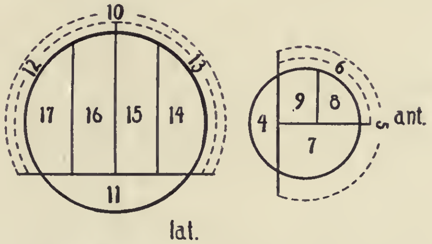


Fig. 12.—Diagram of the segments into which the sciatic nerve was split in the electric stimulation experiment. The numbers of the segments correspond to the tracing numbers in Figures 13 and 14; for example, when segment 4 (above) was stimulated, the record of the muscles which contracted is that in Figure 13, tracing No. 4.

With a fine cataract knife the nerve was then split for about 1.5 cm. into its two divisions which were grossly visible: the common peroneal and the tibial. The first was then stimulated. The tracing obtained is shown in Figure 13, No. 2. The muscles supplied by the common peroneal contracted while those supplied by the tibial did not. Then the tibial was stimulated. The four tibial muscles contracted; the peroneal group did not.

Next the common peroneal division was split longitudinally for about 1.5 cm. into a number of segments as diagrammed in Figure 12. When segment No. 4 was stimulated all the muscles of the peroneal group contracted; those supplied by the tibial did not (Fig. 13, No. 4). When the remaining portion of the common peroneal was stimulated (Fig. 12, No. 5) all the muscles of the peroneal group again gave a curve, while those of the tibial remained unaffected (Fig. 13, No. 5). The same was true for stimulation of all the other segments into which the common peroneal nerve was divided. This is easily seen by comparing the segment number in Figure 12 with the corresponding tracing in Figure 13.

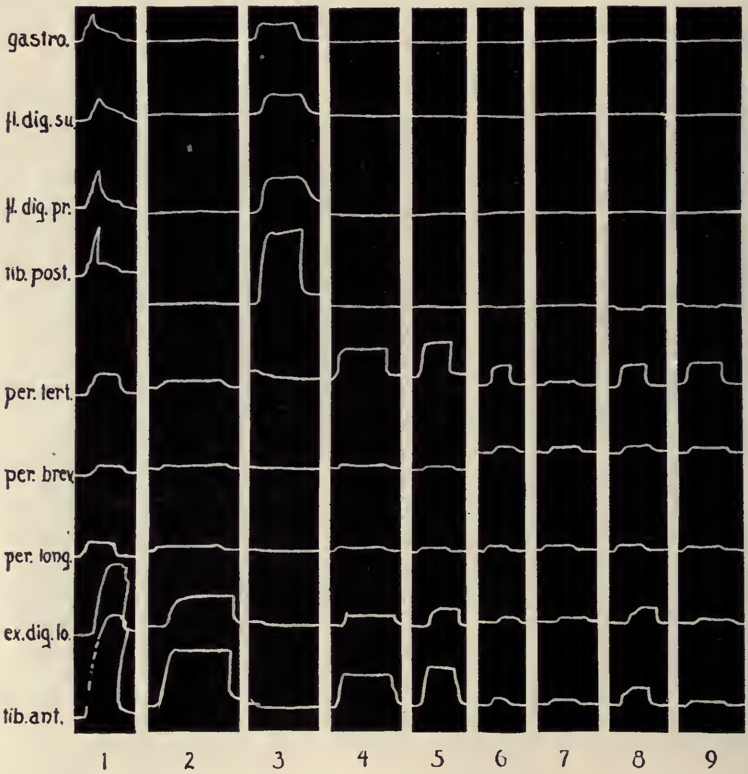


Fig. 13.—Tracings of the muscle contractions in the electric stimulation experiment.

The tibial division was treated in the same way. Here also the tracing number in Figure 14 corresponds to the segment number in Figure 12, which shows diagrammatically the approximate segments into which the tibial was split and then stimulated. In no instance was there contraction of the muscles supplied by the common peroneal.

In every case, except in the stimulation of segments 11 and 14, there was contraction of all four of the muscles of the tibial group from which tracings were obtained. On stimulation of segments 11 and 14 only the flexor digitorum profundus and the tibialis posterior contracted

We repeat that the current used was the minimal to produce muscle contraction in order to avoid overflow.

This experiment indicates that in the sciatic of the dog at the level between the ischial tuberosity and the greater trochanter of the femur there is little grouping of the motor fibers into functional groups aside from the separation of the nerve into its two main divisions. The observations would have been more satisfactory if they had been carried out on a species whose sciatic is made up of a number of grossly visible fasciculi, as in the human being.

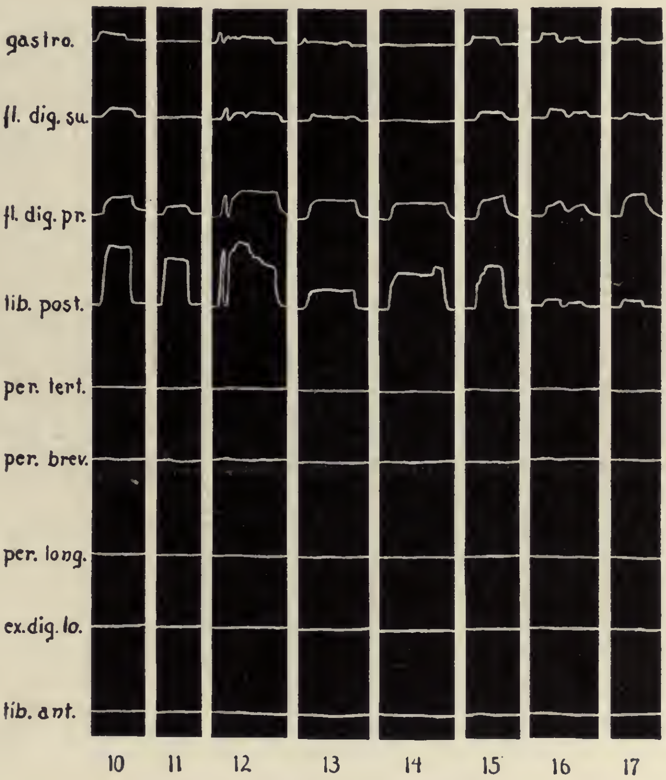


Fig. 14—Continuation of Figure 13.

DISCUSSION

It has been shown in the foregoing that the tibial and common peroneal divisions of the human sciatic nerve should be considered as vast plexuses of nerve fibers from the sacral plexus to at least the distal part of the proximal third of the leg (well into the common peroneal and tibial trunks proper). This continuous exchange of fibers among the various fasciculi making up the nerve can mean only a continuous modification of the functional topography of the nerve throughout its length and definitely negatives the conception of Stoffel,[2] Marie,[3] and Kraus and Ingham,[5] who would have us think of fasciculi in terms of peripheral branches. The only true functional localization in the sciatic, aside from separation into the tibial and common peroneal divisions, is that seen just above the points of departure of branches, since the fasciculi to the branches may be traced along the nerve trunk for an average distance of 6.5 cm. (maximum 21 cm., minimum 1 cm.) before they are lost among neighboring bundles of nerve fibers.

In conformity with this we find, in the dog, that partial division of the sciatic between the ischial tuberosity and the greater trochanter of the femur shows complete separation of the nerve into its tibial and common peroneal divisions, but that partial division of either one of these two causes degeneration, not in single branches but in many of the respective branches of each of them. Hence, there must be very definite mixing of the nerve fibers throughout their course from the sacral plexus and the tibial and common peroneal nerves before peripheral branches are given off.

Electrical stimulation of various sections of the sciatic in the dog at the before mentioned level provokes contraction in all the muscles supplied by either the tibial or common peroneal, depending on which one is stimulated, and not in one or two of the muscles (exception noted in the foregoing).

Thus, in the light of this investigation and that of Dustin,[10] it is impossible to conceive of any functional significance of fasciculi in peripheral nerves in the sense that "the course (of motor fasciculi) is a straight one from the point where the nerve has been made up by its contributing segments to the point of offset of the fasciculi as a branch"—Kraus and Ingham.[5]

In case of a transverse lesion of a nerve it is customary to operate as soon as one is convinced that there is no infection in the field of operation and no evidence of regeneration of the nerve—U. S. Army Manual.[6] The operation then consists in resecting the scar tissue, the neuroma of the central stump and the glioma of the peripheral stump, until the normal nerve fasciculi in the one and the atrophied bundles of the other are distinctly evident. In the average operation, two or three or more centimeters of the nerve must be resected. Then it is advised to suture the divided ends without torsion in as nearly their normal anatomic relationships as possible. Marie says: "It is indispensable that in their sutures, the surgeons appose as exactly as possible the sectioned ends of the various divided nerve fasciculi of the superior end with the same fasciculi of the inferior end." The impossibility of following to the letter any such procedure is at once apparent. Reference to Figure 6 shows that within 2 cm. of the length of the sciatic trunk there are twenty-three fusions and fifteen divisions of the various bundles at this point and a reduction of eight fasciculi. Thus, after resection of the glioma and neuroma, the fasciculi in the proximal stump cannot correspond to those in the peripheral stump. This, however, does not mean that we cannot support the point of view of a minimum disturbance of the nerve topography in the way of torsion.

As Dustin[10] points out, success of the suture of a nerve depends to a decided extent on the number of fasciculi in the nerve at the point at which it has been divided. If the area involved is where the nerve consists of many bundles, the growth of fibrous connective tissue from the perineurium between the two stumps is much more likely, apposition of normal fasciculi in the proximal stump to those in the peripheral stump becomes much more a matter of chance, and the regenerating axons are much more likely to go astray than if the section has occurred through a "nodal zone" (Dustin) where the fasciculi are few and relatively large, and where there is a minimum amount of connective tissue within the nerve proper. Though no statistics are available, one can deduce from the foregoing that, other things being equal, the prognosis of a sciatic lesion producing complete anatomic interruption is better in direct proportion as the lesion approaches the level of the knee where the fasciculi are few and large.

Again, on the basis of anatomic considerations, it is evident that partial division of the sciatic nerve just above the point of departure of a peripheral branch would be more likely to give symptoms of division of the branch than if the lesion were located higher up; in other words, there is localization of function anatomically for the various branches for only a few centimeters proximal to the level at which they leave the nerve. Hence, partial division of the nerve trunk would probably be more serious as regards a particular branch in direct proportion as the level of the lesion approaches the point of departure of the branch. Sherren[7] has already noted this principle as a clinical fact without explaining the underlying reasons for it.

HEMIHYPERTROPHY AND MENTAL DEFECT

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Anomalies of anatomic development and mental defect are often found in close association. A noteworthy example of this fact came to the writer's attention in 1913. A boy, then 13 years of age, was presented for mental examination. He was found to be mentally deficient. A physical inspection of the whole body of the boy showed this deficiency to be associated with one of the most remarkable of developmental anomalies; namely, total unilateral hypertrophy.

This anomaly is considered to be one of the rarest in the domain of medicine. Marked cases are indeed extremely few as a survey of the literature will reveal; but how frequent mild, unrecognized cases are, remains to be determined.

It is because hemihypertrophy presents a startling, though legitimate, deviation from an almost universal law of development that it constitutes a subject rich in suggestion—and perplexity.

A brief description of the physical and mental characteristics of the case here reported will serve as a basis for the discussion of this interesting condition.

REPORT OF AUTHOR'S CASE

Physical Development.—The boy in stature was fairly well developed for his age. At 20 years of age he was 67 inches tall and 137 pounds in weight. His height-weight index at the age of 13 was also normal, being then 87 pounds for 57⅝ inches. He was phlegmatic in his movements and of moderate endurance. He was one of eight children. The brothers and sisters were average or normal, both physically and mentally. His parents were likewise normal and no facts of hereditary significance could be gained.

The birth was natural, without instrumental assistance, and the mother nursed her child into the second year. Dentition apparently was not delayed.

The mother asserted that she had noticed larger development on the right side in early infancy, and there can be no doubt that the condition was congenital. No children's diseases or accidents of significance were to be noted.

The accompanying tables of measures and illustrations indicate the character and degree of the hemihypertrophy. The first table indicates the physical, and incidentally the mental, status of the subject at the age of approximately 13 years. Seven years later, after adolescence had been practically completed, similar measurements were made and these are presented for comparison. They indicate that the condition of hemihypertrophy is, at least in this case, a relatively stationary one, exhibiting neither a progressive nor ameliorative tendency.

TABLE 1.—Table of Measurements (Age 13)

Height, 57⅝ inches.

Sitting height, 29¼ inches.

Weight, 87 lbs. Head: length, 18.2 inches; breadth, 15.4 inches; circumference, 56.5 inches.

Dynamometer: right, 15; left, 11.

Chronologic age 12½ years; mental age 4½ years; intelligence quotient 36.

	Right	Left	Difference
Arm, length	63	61	2
Forearm, length	38	36	2
Forearm, girth	18	17	1
Wrist	14	13	1
Hand	21	18.5	2.5
Middle finger, girth	7	6.5	0.5
Middle finger, length	10	9	1
Metacarpal bone III, length	46 mm	43 mm	3 mm
Capitale bone, length	20 mm	17 mm	3 mm
Leg, (A. s/s/. to ex. mal.)	80	75	5
Leg, perin. to in. mal.	63	61	2
Leg, patellar edge to in. mal.	34	30	4
Thigh girth	39	38	1
Knee girth	30	29	1
Calf girth	29	28	1
Foot, metatarsal bone, length	54 mm	52 mm	2 mm
Metatarsal bone, width of head	18 mm	15 mm	3 mm
Ex. canthus to antetragus	8	7.5	0.5
Int. canthus to nasolabial junction	5.2	4.5	0.7
Ear, length	7.5	6.5	1
Lip, upper	4	3	1
Upper canine (width)	10 mm	9 mm	1 mm

Examination of the blood and urine gave negative results. The red blood cells numbered 5,424,000; hemoglobin, 95 per cent.; white blood cells, 10,000; polymorphic cells, 68 per cent.; large mononuclears, 21 per cent.; small mononuclears, 11 per cent.

TABLE 2.—Table of Remeasurements (Age 20)

Height, 67 inches.

Sitting height, 33¾ inches.

Weight, 137 lbs. Head: length, 18.7 inches; breadth, 15.9 inches; circumference 59 inches.

Dynamometer: right 31; left, 22.

Chronologic age, 20 years; mental age, 5 years, 8 months; intelligence quotient, 35.

Anthropometric Measures	Right	Left	Difference
Arm, length	77.5	75	2.5
Forearm, length	46	44.5	2.5
Forearm, girth	25	23	2
Wrist	16.5	14.5	2
Hand	23	20	3
Middle finger, girth	7.5	7	0.5
Middle finger, length	12.5	11.5	1
Cranium: segments of intertragal arc	19	17	2

The axillary temperature was equal on both sides.

The asymmetry was not very marked. It might easily escape observation, though it caused the boy to limp to a moderate extent.

Careful inspection revealed mild gigantism involving apparently all of the right side: the right ear was longer, the right half of the nose larger, the right nares twice that of the left in diameter, the right palpebral fissure was wider; on the same side the cheek and lips were fuller; the arm was longer, the right hand was relatively more enlarged than some of the other structures; the right leg and foot were similarly enlarged; the penis was definitely enlarged to the right of the median line.

The opthalmoscope showed no marked difference in the right fundus, though the veins were fuller. There were no differences in refraction in the two sides.

Throat examination revealed no marked difference in the tonsils. The uvula was bifid; the epiglottis was definitely asymmetrical, but curiously enough, the left side was thicker than the right; the posterior rim was also bulkier on the left side, the whole right of the epiglottis apparently being in the infantile condition.

A cast (Fig. 5) of the upper palate and teeth showed considerable deformity. The placement of the teeth was very defective on the right side giving the gums a thicker appearance on that side. The right upper canine was 10 mm. wide and 9 mm. thick; the left canine was 9 mm. wide and 8 mm. thick. The eruption was further advanced on the left side, which is contrary to rule.

The skin was by no means normal. The peculiarities, presently to be noted, were more pronounced on the right side but were by no means limited to that side. The skin was dry and there was a definite tendency to ichthyosis. Small keratinous patches were present about the elbows and the ankles. Most prominent and characteristic were extensive areas of telangiectatic congestion which gave the skin a marbled, and, in some areas, a decidedly livid aspect; for example, the dorsal surface of the right hand and the lateral surface of the leg. On last examination the right leg had developed marked varicosities (Fig. 4).

On palpation the hypertrophied side had a more doughy feel than the left. This suggested redundancy of the subcutaneous tissue, but the roentgen rays showed that the bones themselves had participated in the hypertrophy. The measurements showed that all the members of the right side were not proportionately hypertrophied.

Mental Development.—The mental development of this boy has shown a consistent retardation dating no doubt from birth. He was over 2 years old when he began to walk; he began to say "mama" and "papa" at the age of 3; but he did not begin to "talk" until about the age of 5. Even now he speaks in phrases and short sentences and has a very childish vocabulary. He entered the first grade of the public schools at the age of 9. He stayed in this grade two years and was gradually promoted into the higher grades out of deference to his physical development. His academic attainments after years of schooling never rose to those of grade 2. During all these years he has been considered a docile, amenable, cheerful and neat boy. He has been able to read a few words, to spell a few others and to repeat a few number combinations. Persistent drill has left some precipitate, but there has been a restricted intellectual development during the seven years of teens in which I have observed him. He is now 20 years old and, although his intelligence is more mature than it was at the age of 12½, he still must be classified as an imbecile.

At the chronologic age of a little less than 13, he had a mental age of 4½ years by the Vineland revision of the Binet tests. At that time he could not tell which was the longer of two lines, one 7, and the other 6 cm. in length; he was also unable to repeat a sentence consisting of ten words. In another year he was able to meet both of these tests, but otherwise he showed little progress. After six more years of growth, he is still unable to distinguish between right and left and cannot tell whether it is morning or afternoon; but he is able to count 13 pennies, and he can describe a simple picture. The net result is that now at the age of 20 he has a mental age of 5 years and 8 months. In other words, it has taken about seven years for this boy to accomplish the equivalent of one year of mental growth. This growth has been as steady and consistent as his physical growth; but it has been slower and on a different level. There has been no deterioration. Seven years ago the ratio between his mental age and chronologic age was 4½ : 12½, which expressed on a percentage basis gives an intelligence index or quotient of 36. By a similar method of calculation, using the Stanford revision of the Binet scale, we find that his present intelligence quotient (IQ) is 35. Though these two quotients were not derived by precisely the same method, they are comparable and true to the facts of the case. We cannot say that this youth has shown no mental growth during his teens; but viewed in relative terms, we are obliged to conclude that there was no increase in his caliber. His IQ has remained fairly constant.

His actual mental status is summarized in the accompanying normgraph (Fig. 8), which in terms of age norms, plots his ratings. In height and weight he was, and is, at standard; but in the psychophysical measurements obtained by the dynamometer and spirometer, and in the mental and educational measures he is at the level of a beginner at school. On this level he will remain. At the age of 20 his drawings of a house, man and bird are characteristic of a kindergarten mentality.

It would be unjust, however, to leave this sketch without referring to the surprising success which this boy has made in life in spite of his limitations. His mother, though a struggling widow with eight children, was loathe to consider commitment to an institution. We respected this attitude and repeated efforts were made to find a simple occupation for him. There were great difficulties as employers would not take the necessary risk, pains and patience. Finally, the cooperation of neighbors was enlisted and he got a job as a scullery boy at a downtown café, with hours from 8 to 5 and wages of $5.00 a week. For a whole year he has been doing regular, satisfactory work, the pride of his mother. And incidentally this is an example of the sort of thing that local communities might deliberately do to control the problem of feeblemindedness.

SURVEY OF RECORDED CASES OF HEMIHYPERTROPHY

Thomas Wedders, with a nose reputed to be 7½ inches long, was a famous character in an earlier day. The scientific literature on the subject of hypertrophy, however, goes back to the publication of a case by Wagner in Germany, in 1839, and one by Desvouges in France in 1856, and by Adams in England in 1858. Osler, writing in 1879, gave the number of cases of congenital hypertrophy reported to date as twelve. Ely Leblanc, who in 1897 wrote his medical thesis on the subject, made an exhaustive canvass of the literature and

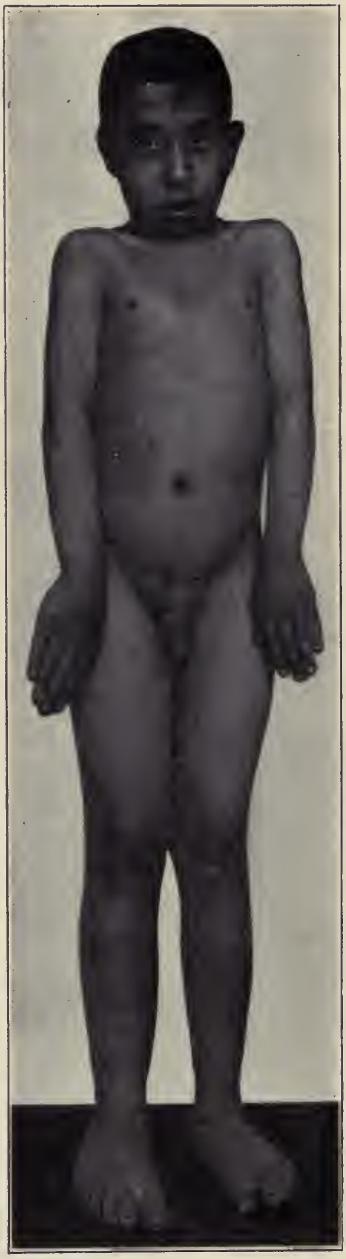

Fig. 1.—Hemihypertrophy; subject aged 13.

assembled seventy-nine case, of which twelve were cases of total hemihypertrophy, thirty-eight of partial, nineteen involved but one extremity, six the face and four were crossed. Lewin assembled sixty cases of hypertrophy limited to fingers and toes. David M. Greig, in 1898, in the Edinburgh Hospital Reports reviewed a collection of forty-two cases. This collection was confessedly incomplete and included a large proportion of cases of partial hypertrophy as distinguished from total unilateral hypertrophy or true hemihypertrophy. Greig's valuable monograph deals chiefly with unilateral cephalomegaly (unilateral hypertrophy limited to the head and face), of which he collected thirty-five cases. I am indebted to Greig and Leblanc for references to several cases considered below.

I was able to collect forty cases of true hemihypertrophy, and this may be regarded as a reasonably complete list of the cases now available in the medical literature. Table 3 gives a list of these cases. For comparative purposes a tabulation of thirty cases of partial and crossed hypertrophies is included. Table 4 is by no means complete, for such partial or restricted hypertrophies are much more common than true total hemihypertrophy. It is not unlikely that they frequently have a pathology and etiology identical with that of hemihypertrophy. We shall, however, limit our discussion to the latter condition.

An analysis of Table 3 shows that of forty reported cases only five patients were adults, nineteen were male, seventeen female, and four not specified as to sex. In twenty-seven, or almost 70 per cent. of the cases, the total unilateral hypertrophy was on the right side. For every case of left-sided hemihypertrophy there are two right-sided cases. In fifteen of the cases it was definitely stated that all the tissues were involved in the hypertrophy. In nineteen cases abnormalities of the skin were mentioned as a complication, while in three cases the skin was reported as normal; in the remaining number no facts on this point were given. The data as to mental status are incomplete. In twenty-nine cases no facts are given; in many of the instances, indeed, the subject was too young to make the mental status obvious. In only six cases was the mentality reported as normal and in five cases, or 13 per cent. of the total, it was reported as defective.

A general statistical summary of the facts in Table 4 shows a similar picture. Only three of the listed cases of partial hypertrophy occurred in adults; males were slightly more numerous (fourteen to eleven); the right side was more frequently involved than the left in the ratio of 16:9. In five cases, however, both sides were partially involved in the same person. Skin complications were reported in eleven cases; in only one case were they recorded as absent. Mentality

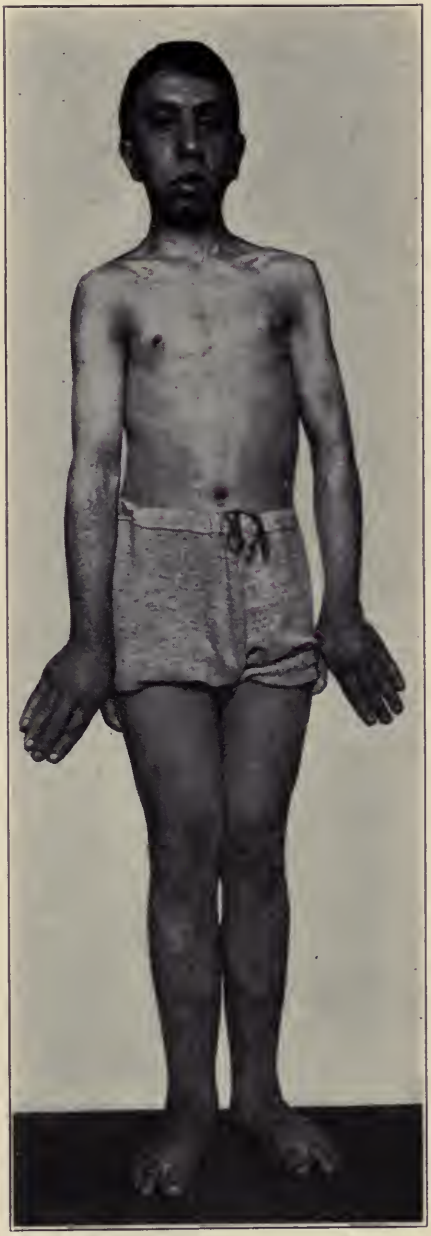

Fig. 2.—Same subject as in Figure 1, aged 20.

TABLE 3.—Cases of Total Hemihypertrophy

Author	Sex	Age	Side	Mentality	Skin Complications	Special Features
Adams, S. S.	M	10	Right	Normal	Telangiectasis	Hair darker and longer on right side
Ahlfeld	F	2	Left			
Arnheim	F	2	Right	....	Nevuspilosus hypertrichosis	Right teeth adult in size; right kidney 1 cm. longer; tongue asymmetric; necropsy
Bassoe	M	16	Left	....	Nevi pigmented hypertrichosis	
Broca	M	14	Left	Subnormal		
Brüning	M	1	Right	....	Nevi pigmented angioma	Hypertrophy of 2 fingers, left side
Bull	F	..	Left			
Burlet	F	27	Right	Subnormal	Vascularity increased	Temperature + on right side
Caglati	F	15 mo.	Left	....	Nevi pigmented keratosis	
Demme	F	1 wk.	Left	....	....	Necropsy: left cerebral hemisphere +
Desvouges	M	17	Right	....	Nevi; varices	2 left toes +: right side stronger
Eaton	M	10	Left			
Fatichi	F	53	Left			
Finlayson	F	18 mo.	Right	....	Dark red on right side	Skin on right side blue on crying
Fortescue-Brickdale	M	9 mo.	Right	Normal	Nevi	
Fischer, H.	F	9	Right	....	Venous ectasia	Right side more fatigable
Fowler	F	10 mo.	Right	....	Nevi	
Gesell	M	13	Right	Subnormal	Telangiectatic patches	Reversed asymmetry of epiglottis
Gordinier	M	65	Left	Normal	Nevi	Necropsy: left cerebral hemisphere +
Hornstein	F	2 mo.	Right	....	Nevi	Necropsy: connective tissue hyperplasia
Hutchinson	M	5	Right	....	....	Necropsy: suprarenal, testicle, tonsil, etc.
Jacobi	..	3 mo.	Right			
Langlet	..	Adult	Right			
Logan	..	4	Right			
Masmejean	..	1	Right			
McGregor	M	10	Right	....	....	Necropsy: right optic thalamus +
Milne	F	19 mo.	Right	....	....	6 teeth on right side; 1 on left side
Milne	F	12	Right	....	Telangiectasis	Right forefinger and thumb normal
Monod	M	19	Right	....	Telangiectatic varicosities	Temperature + on right side
Möbius	M	14	Left	....	Telangiectatic marbling	Pains in left arm
Mummery	M	5	Left	Normal	....	Left side apparently grew faster in 2 years
Ollier	F	27	Right	Subnormal	....	Temperature + on right side; cephalalgia
Pollosson	M	Adult	Right	....	....	Elephantiasis (?)
Redard	M	2	Right	Normal	Nevi	Hypertrophy right foot very marked; temperature right side +
Schiller	M	11	Left			
Telford	F	7	Right	....	Nevi, varicosities	Rickets equal on both sides
Thomson	F	18 mo.	Right	Subnormal	....	Thyroid absent? cretinism?
Tilanus	F	10	Right	Normal	Telangiectatic marbling	Right side stronger
Turbil	M	2 wk.	Left			
Wagner	M	11	Left	....	Telangiectasis: blue pigment	

was reported normal in five cases and abnormal in three, the subnormality in two instances being associated with marked cranial asymmetry.

Of peculiar interest is the relatively frequent association of mental defect and hemihypertrophy. In 13 per cent. of the cases in the series this association is present. If the mental status could have been determined in every instance, it is possible that the proportion might have been markedly increased. The facts clearly indicate that hemihypertrophy should be added to the list of developmental anomalies which bear some lawful relation to the incidence of mental deficiency. What is the nature of that relation?

TABLE 4.—CASES OF PARTIAL HYPERTROPHY

Author	Sex	Age	Side	Mentality	Skin Complications	Special Features
Adams	M	19	Right	....	Nevus	Leg on right side 1½ inch longer
Adler	..	..	Right	....	....	Foot
Ballance	M	17	Left	Subnormal	....	Head and certain toes of each foot
Bankhart	M	6	Right, left	....	....	Right side of face, tongue leg +; left arm +
Beck	M	28	Right	....	....	Thumb, forefinger, arm, right tactility —
Chassaignac	M	18	Right	....	Varices	Right arm, leg
Chassaignac	F	..	Left	....	Nevus	Leg only
Cohem	M	56	Right	....	....	Right leg: sugar tolerance +
Curling	F	..	Right, left	....	....	Right middle, ring finger 5½ inches; left thumb +
Davidson	F	1 mo.	Right	....	....	Leg
Foucher	M	..	Right, left	....	Venous hyperemia	Left thorax, abdomen +; right fingers +
Friedberg	F	10	Right, left	....	Telangiectasis, pemphigus, elephantiasis	Right leg adult size, right foot ++
Friedreich	F	16	Right	....	Pigmentation; abnormal hair	Head: saliva + on right side, taste + on left side
Goldscheider	M	19	Left	....	....	Thumb, forefinger; sensation equal
Gruber	M	20	Left	....	....	Enormous hypertrophy of forefinger (16.5 cm.)
Hutchison	..	4 mo.	Left	....	....	Necropsy: left thymus +; left kidney 56 G., right 28 G.
Jacobson	F	3	Right, left	....	Telangiectasis; abnormal hair	Right cheek, arm, hand +; left breast, abdomen, leg +
Lewin	M	26	Right, left	....	Nevi; varicosities	Left side of face, arm, temperature, sweating +; right leg +
Osler	F	9	Right	....	....	Right arm adult size
Maschke	F	5	Left	Normal	....	Synostosis of proximal phalanges
McConnell	..	7 mo.	Left	Normal		
Passauer	M	11	Left	Normal	....	Marked facial asymmetry left cheek, tongue, teeth +
Pershing	F	19	Right	....	....	Trophedema ?
Prince	F	4 mo.	Right	....	....	Forearm, forefinger, thumb ++
Redard	M	3	Right	....	....	Temperature 2 to 4 degrees higher on right side
Reissmann	M	7 mo.	Right	....	....	Left cranium +; left dentition retarded
Speese	F	10	Right	....	....	Toe enormous size
Steffen	..	11 wk.	Right	....	....	Necropsy: hyperplasia; right hemisphere +
Widenmann	M	10	Right	Subnormal	Abnormal pigmentation	Right side of cranium +
Wittelshöfer	..	..	Right	....	....	Second, third toes +

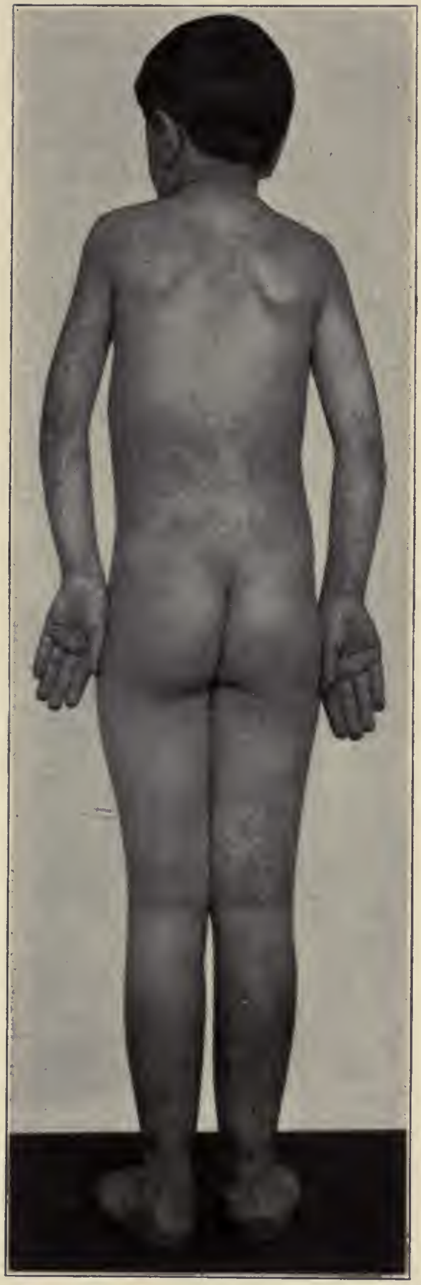

Fig. 3.—Posterior view of subject in Figure 1, aged 13.

THE ETIOLOGY OF HEMIHYPERTROPHY

Since we do not know the true cause of hemihypertrophy, there is no dearth of etiologic theories. These "explanations" range from maternal impressions to internal secretions. A German mother ascribed the hemihypertrophy in her son to the fact that during pregnancy she had seen a youth of gigantic proportions in the market place. Presumably she saw only one side of this giant.

One writer attributes hemihypertrophy to an inherent tendency of the tissues to appropriate an excess of nutriment, but, as Ballantyne remarks, this statement is more ingenuous than ingenious.

Fortescue-Brickdale advances a venturesome biochemical theory. He suggests that the condition is due to an irregular distribution in the tissues of the anchoring substance which interacts with the glandular secretions which determine growth.

Another group of theories ascribes the hypertrophy to vascular lesions, calling attention to the frequency of vascular and skin complications and to the analogy of "naevus neuroticus unius lateris." One view is that an embryonic disturbance establishes a preeminence in the blood supply of one side or one part. Once established, this preeminence causes a permanent overgrowth of the favored side. This explanation is similar to that which ascribes right-handedness to the richer blood supply of the left hemisphere. Partial vasomotor paralysis has been suggested (Virchow); also incomplete development of the middle arterial coat. Pollosson regards a disturbance of the lymphatic system as the etiologic factor. But if the lesion is a vascular one, what is its origin? Is it hereditary?

There is no evidence that hemihypertrophy is hereditary. Bruns found only one case in the literature in which macrodactylism was present in several members of a family. There is one record of a family with apparently hereditary enlargement of the left hand, but we know of no instance in which two members of the same family or related members presented hemihypertrophy. To be hereditary the differential cause of a character must be present in the germ cell. Hemihypertrophy is congenital, but in origin it is probably embryonal and not germinal.

Hemihypertrophy may easily date back to some disturbance during the early embryonic period. The results of experimental teratology have shown that gravity, mechanical and other influences may induce monstrosities of various kinds. It is conceivable that a slight deviation from the harmony of physical influences surrounding the embryo may be competent to initiate a partial or unilateral hypertrophy in ways similar to those demonstrated in the laboratory. If the influence is teratologic, it may well go back as early as the fourth week in the embryonic period during the cleavage of the mesoblastic somites.

Wieland therefore regards hemihypertrophy as an embryonal malformation: "By continuous or intermittent proliferation processes in these somites during the intra-uterine life, the more severe types of congenital hypertrophy arise."

Leblanc similarly traces the lesion to the primitive embryonic neurotomes and their associated vasomotor center.

Greig takes the same general view and makes it more explicit. He argues that in the embryo all motor nerves may be regarded as trophic, because if they are destroyed or injured the parts supplied by them are more or less abnormally developed. Therefore it is to a central lesion or irritation of certain nerves brought about by intra-uterine meningitis or cerebritis that one must look for the origin of unilateral hypertrophy. Pathologic evidence adduced to support this interpretation is the enlargement of the foramina of certain nerves suggesting a previous inflammatory process.

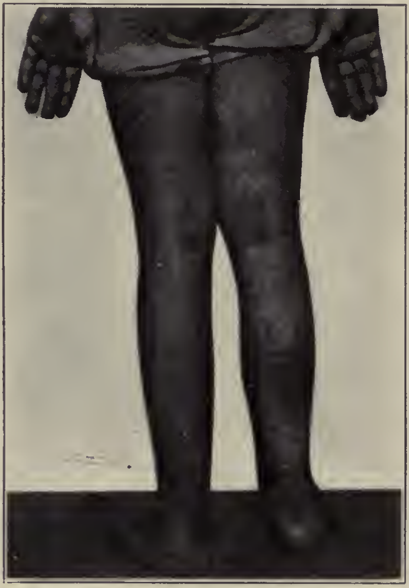

Fig. 4.—Posterior view of right leg showing areas of cutaneous congestion; subject aged 20.

If it is impossible to establish the existence of such definite extrinsic causes operating in the embryonic period, it will be necessary to search so far as we can among the deeper determinations of growth and development.

Growth depends on two groups of factors—internal and external. In the latter group are included the activators, hormones, vitamines, food accessories and secretions. The action of these agencies, however, is general and mediated through the general blood supply. It is difficult to conceive how that action could be diminished or increased in a restricted manner in one member like the middle finger, or one side of the whole body to the exclusion of the other. Among the internal factors of growth are the so-called determiners which are already laid down in the fertilized ovum. It may be that for all paired or bipartite organs, instead of one determiner, there are two, or two groups of determiners, and the basis of asymmetry depends in some unknown way on an inequality of these primary determiners.

One investigator considered that in his case of hemihypertrophy the enlarged side looked fully a year older than the other, and that the ossification at the epiphyses of the forearm was, on that side, more advanced. Ossification is a criterion of physiologic age. A man is as old as his arteries, and a child, if we follow Dr. Rotch, is as old as the bones of his carpus. Does this mean that due to the difference in growth factors our boy with hemihypertrophy has three ages: his chronologic age, a right physiologic age and a left physiologic age? The fact that detention on the hypertrophied side is often precocious to the extent that there will be eight teeth on that side and none on the other is not altogether inconsistent with this fancy.

TWINNING AND ASYMMETRY

It is natural that a discussion of the etiology of hemihypertrophy should finally bring us to problems of double physical personality and twins. Further researches into the biology of twinning may bring the remarkable phenomenon of unilateral hypertrophy more completely within our comprehension; may even prove it to be on closer scrutiny more frequent and less anomalous than we had supposed. Indeed, even now, all things considered, the real marvel is not the occurrence of hypertrophy but the fact that hemihypertrophy is such an extreme rarity.

"By twinning we mean production of equivalent structures by division." This statement is taken from the biologist Bateson, who regards the power to divide as a fundamental attribute of life. The tendency to symmetry, to bilateral equivalence or mirror imaging is so general that it also must be regarded as a fundamental of biologic mechanics. Hemihypertrophy accordingly may be conceived as some profound inaccuracy in the natural process of developmental duplicity. It is not as monstrous as the double monster, but it may have a related morphogenesis. At any rate, we can safely assume that hemihypertrophy is not an artifact really consisting in a hemi-atrophy. It is evidently a mild unilateral gigantism of an individual whose lesser somatic half is normal.

In a certain biologic sense we may regard every bilateral individual as being a pair of twins. H. H. Newman, in his fascinating work on the "Biology of Twins," holds that monozygotic twinning—where a single egg produces two offsprings—is "a phenomenon that should be considered as only a phase of the much more general phenomenon of symmetrical division. The development of the right and left hand homologous organs in a bilateral organism is essentially a twinning process." This author also observes that "the whole matter of bilateral development appears to be quantitative in nature, in that the same type of process may go not so far or further than normal." Just as there may be an inhibition of the normal culmination of the process of bilateral division (as in the single median cyclopic age), so there is frequently an excess of division resulting in the two bilateral structures becoming completely segregated, as when a single individual develops two heads or two tails, while the remainder of the organism is a more or less normal individual. Newman, like Bateson, regards the phenomenon of twinning as a fundamental process which is almost universal in the field of biology.

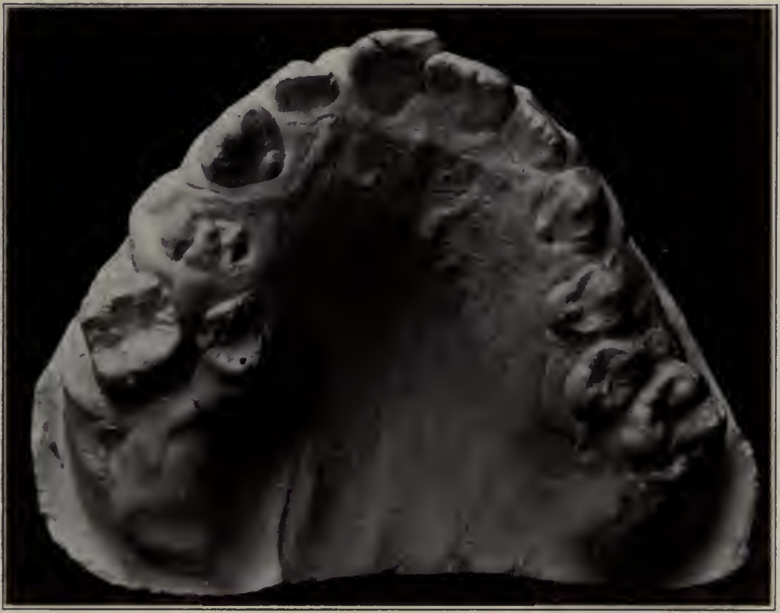

Fig. 5.—Cast of palate and upper teeth.

From this point of view, hemihypertrophy may be interpreted as an atypical or imperfect form of twinning, a variant of the same process which may produce a double-headed monster or a perfectly, ordinary normal individual—an ordinary individual being an organism in whom there has been a precisely balanced inhibition on the biologic process of bilateral doubling.

The actual mechanism of this regulative control over symmetry of size and form remains somewhat enigmatic in spite of much biologic investigation. Davenport regards size or stature as a unit character of inheritance subject to Mendelian principles; but this does not assist us in interpreting the curious stature anomaly embodied in hemihypertrophy.

Newman has made suggestive researches into heredity and organic symmetry in armadillo quadruplets. He has noted some cases in which "one lateral half of the body has quite a different number of scutes from the other half, and one of these halves resembles the maternal condition." Since each set of quadruplets have the same genetic constitution as they arise from one zygote, he concludes that some irregularity in the mechanism of the mitotic cell-division is responsible for the anomalies of symmetry. This factor is by no means a simple one. "Now in the armadillo there are many evidences of a system of symmetry common to all of the quadruplets, upon which has been superimposed a secondary symmetry system between twins. This in twins is more or less obliterated by a tertiary symmetry between the antemeric halves of the single individual."

R. G. Harrison discusses rules of symmetry in his monograph "On Relations of Symmetry in Transplanted Limbs." This study is based on 462 cases of grafting of limb buds in amblystoma punctatum. He agrees with Morgan that the potential factors of symmetry reside in the constitution of the egg. "It is the intimate protoplasmic structure that underlies symmetry." Likewise reversal of symmetry. "As an alternative to the hypothesis of rotation, we might consider reversal as due to reversal of molecular asymmetry according to analogy with the behavior of optically active compounds." "There is an analogy between the production of enantiomorphic limbs and the production of situs inversus viscerum, as effected by Speeman. Either the reversal may be due to reversal of the intimate structure, or it may take place in spite of the intimate structure through the direct action of mechanical factors on the individual parts of the differentiating system." [1]

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[1] Speeman obtained a large number of twins in Triton by constricting the eggs in segmentation stages or in early blastula. In many of the cases one individual, usually the right, developed complete situs inversus viscerum.

Another form of asymmetry, even more startling and drastic than hemihypertrophy is that of gynandromorphism. A gynandromorph is an animal that is male on one side and female on the other. This differentiation may include the reproductive organs, gonads and ducts. Usually it is longitudinally bilateral, but it may be anteroposterior. This curious phenomenon is most frequent in insects but has been reported in birds and in a few mammals. A beautiful case was described in a mutillid wasp in which the male half of the body was black and winged like the male while the female half was a rich red and wingless.

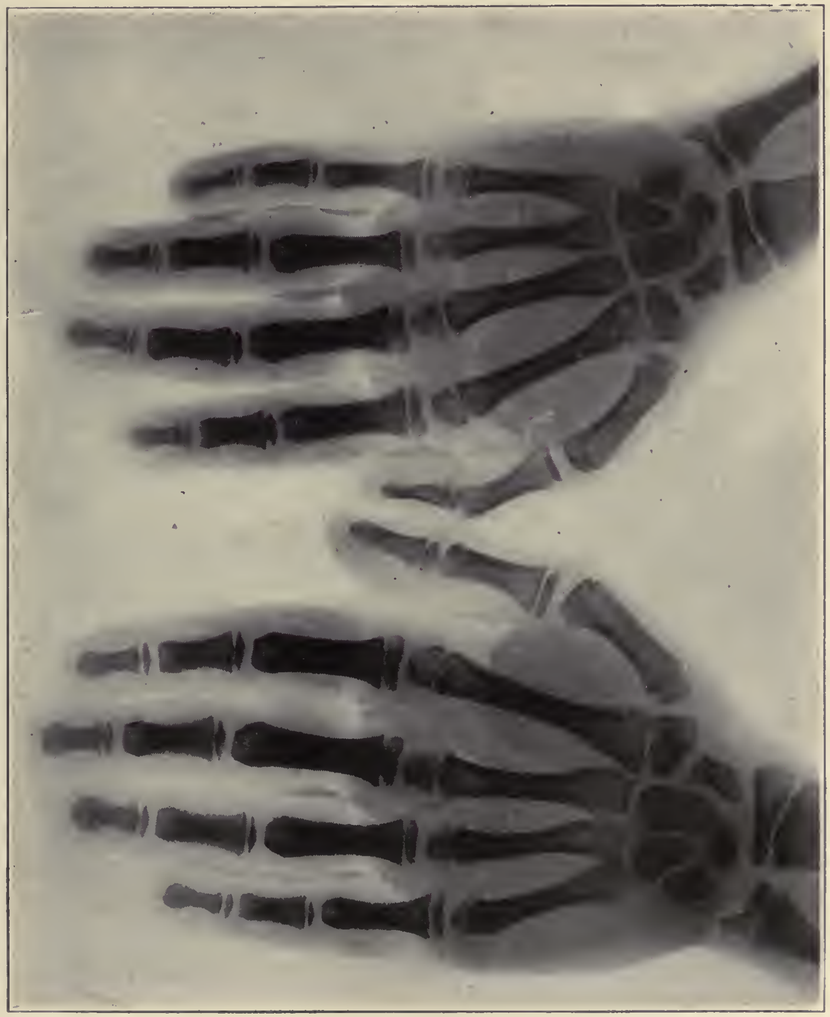

Fig. 6.—Roentgenogram of hands of subject at age of 13.

The problem of gynandromorphism has been extensively studied by F. H. Morgan and C. B. Bridges and reported in their contributions to the genetics of *Drosophila melangaster.* They found one gynandromorph among every 2,200 flies. The authors consider a gynandromorph a hybrid whose genes are carried by the sex chromosome; and they give definite evidence that the peculiar sex mosaic condition is due to an elimination of one X chromosome, usually at some early division of the segmenting nuclei.

MENTAL DEFECT IN RELATION TO HEMIHYPERTROPHY

The foregoing discussion indicates that hemihypertrophy, even though it is frequently associated with mental defect, should not be classified as a stigma of degeneracy. It is congenital, but it is apparently not due to an intrinsic imperfection in the genetic constitution of the germ cells. A stigma of degeneracy would denote such an imperfection. Hemihypertrophy rather denotes an abnormal deviation in morphogenesis and is to be classified as an anomaly of development.

Anomalies of development are found with a high degree of frequency among the feebleminded. Even in growth of stature they show significant departures from the normal. As Goddard found, "low grade idiots have not only a disturbed brain function but their entire organism is disarranged and growth processes upset." In the imbecile the same is true but to a less extent. While mild asymmetry in structure is proverbially normal, pronounced asymmetry is certainly more characteristic of the mentally defective as a class. In a careful study, La Page measured the skull conformation in a series of 198 cases of feeblemindedness and found lateral asymmetry in 158, or 80 per cent., and, what is very interesting in this connection, 122, or 80 per cent. of these cases, showed the hypertrophy to be on the right side. This is in harmony with the ratio between right and left in our series of forty cases of total unilateral hypertrophy. Is it not probable that there is some fundamental similarity or even identity in the causes which produce pronounced cranial asymmetry and hemihypertrophy?

Little is known about the neuropathologic facts. In those cases which have come to necropsy the histologic study of the hypertrophied organs shows that the increase in bulk is due chiefly to an overgrowth of connective tissue. This fact again establishes a possible connection with feeblemindedness. Indeed there is a clinical form of mental defect which is known as hypertrophic amentia, which consists in an overgrowth of interstitial tissue. It may produce a skull, square in shape, 25 inches in circumference. Although this type of amentia is rare, sclerosis (or overgrowth of neuroglia) occurs frequently. Dr. Wilmarth examined the brains of 100 feeblminded children and found evidence of sclerosis in fully one fourth of the cases. Tredgold believes that in some of the cases it is probably due to vascular lesions.

Since hemihypertrophy consists in large measure of a diffuse sclerosis, it may be that we have the production of mental defect in those cases where the sclerosis involves one or both of the hemispheres. One cause would then be adequate to explain both conditions when they are associated.

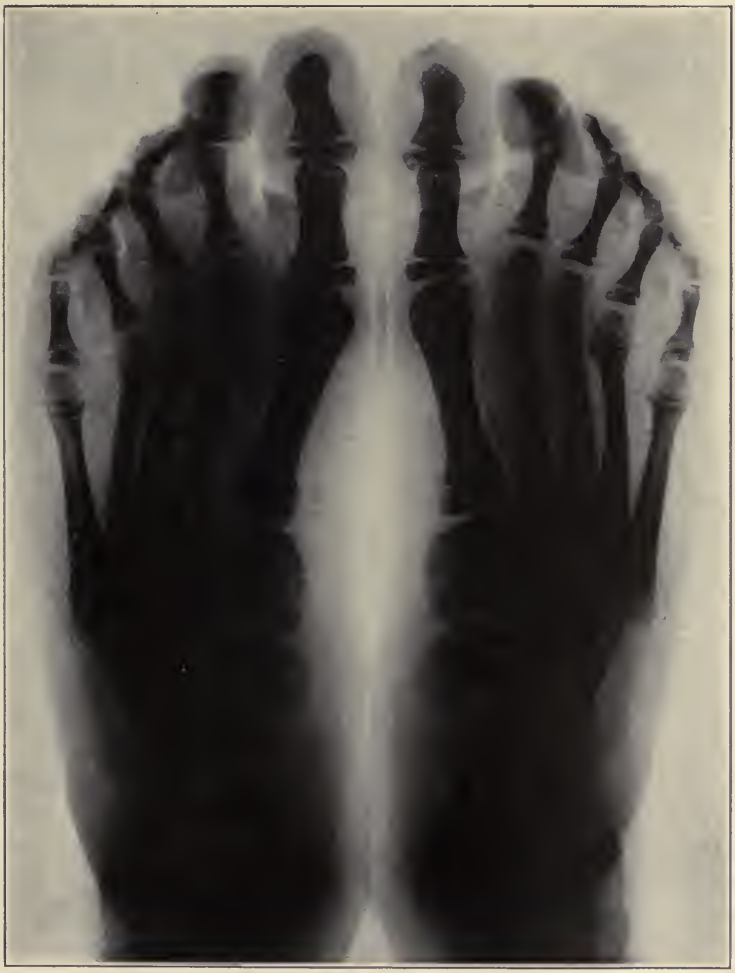

Fig. 7.—Roentgenogram of feet of subject at age of 13.

Furthermore, if the exciting cause of these vascular lesions in the great majority of cases, as Greig suggests, is an intra-uterine meningitis or cerebritis, we have an additional factor for the production of mental defect.

Dr. Gordinier's case of unilateral hypertrophy came to necropsy, but the brain, unfortunately, did not come to microscopic section. The viscera in general showed a diffuse interstitial connective tissue hyperplasia. The left cerebral hemisphere on inspection was distinctly larger than the right, and on measurement proved to be 1 cm. greater in its anteroposterior and 1.25 cm. greater in its transverse diameter. The intelligence of the subject was reported normal.

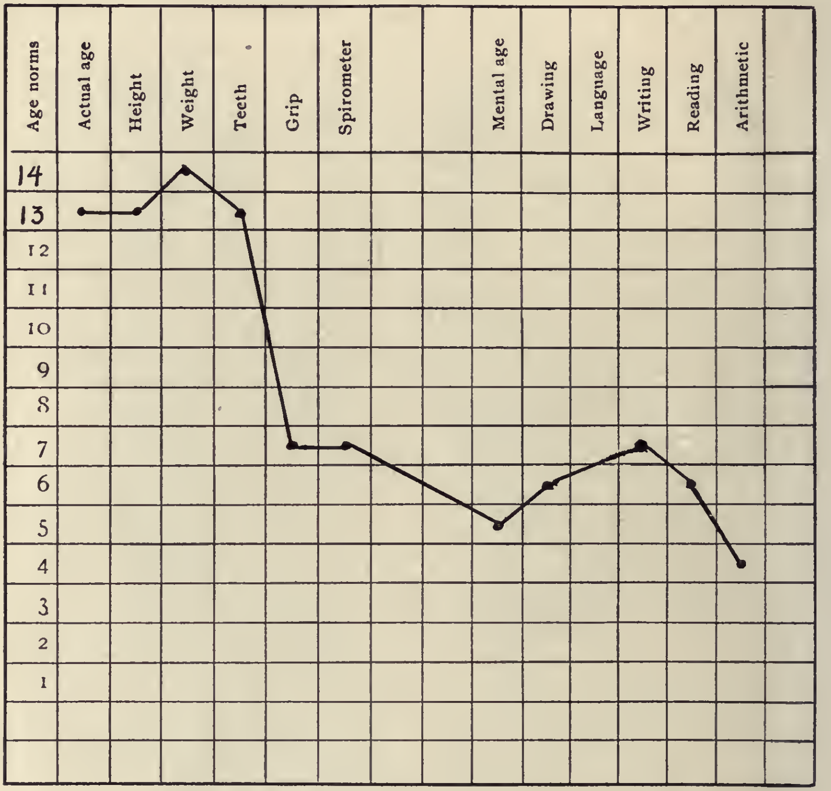

Fig. 8.—"Norm graph" showing standards of physical and mental development.

The production of actual mental defect in any particular case may therefore be contingent on the degree to which neurogliosis has taken place, and to the degree of compensatory development in unaffected nerve tissue.

It is possible that there are different causes for different cases of hemihypertrophy, and it may be that each case represents its own distinctive interaction between some intimate defect in the symmetry determiners and the environment of the multiplying cells. It is tempting, however, to relate true hemihypertrophy and certain not infrequent forms of cranial asymmetry to some quantitative abnormality in the chromosomal mechanism, which produces not only a deviation in the process of bilateral twinning, but also may upset normal tissue relations, increasing the proliferation of certain cells at the expense of others.

This is not out of harmony with Boveri's suggestion that an imperfect or irregular division of the chromosomal complex may, through loss of specific chromosomes, remove regulative inhibitions which permit certain inimical or normally suppressed cells to run wild.

The frequent association of hemihypertrophy and of cranial asymmetry with mental defect and the consistent preference in both conditions for enlargement of the right side suggests some lawful grouping of causative factors. It is quite possible that the origin of certain altogether obscure cases of secondary amentia may lie in an undetectable but decisive imbalance of the fundamental process of twinning which follows fertilization.

Whatever our final hypothesis may be, we seem to be warranted in the broad conclusion that true hemihypertrophy consists in an erroneous deviation from the normal course of development in the embryo, and carries with it, as so many anomalies of development do, the possible penalty of mental defect.

SUMMARY

Hemihypertrophy is a unilateral enlargement of one side of the body, and is one of the rarest among developmental anomalies.

A case associated with mental deficiency is described. Physical and mental measurements were made when the subject was 13 years old and repeated at the age of 20.

A comprehensive tabulation of cases to date (Table 3) shows a total of forty cases recorded in medical literature, of which nineteen were male, twenty-seven on the right side, nineteen with skin complications and five with mental defect.

A less complete tabulation of partial and crossed hypertrophies, including thirty cases, is presented for comparison in Table 4.

In a discussion of the etiologic theories for hemihypertrophy, preference is given to the view that hemihypertrophy is not a hereditary character but a morphogenetic anomaly dating back to an early embryonic stage.

Hemihypertrophy is interpreted as a form of asymmetry due to a possible deviation in the normal process of twinning.

The complication of mental defect is attributed to an abnormality in the process of bilateral twinning which involves a disturbance of normal tissue development.

Possible relations of certain cases of mental defect to cranial asymmetry and intra-uterine meningitis are suggested.

The value of further biometric and clinical studies in asymmetry is indicated.

BIBLIOGRAPHY

Adams, J.: A Singular Case of Hypertrophy of the Right Lower Extremity, Lancet **2**:140, 1858.

Adams, S. S.: A Case of Hemihypertrophy, Arch. Pediat. **2**:901, 1894.

Adler: Ueber partiellen Riesenwuchs, Berl. klin. Wchnschr. **32**:110, 1895.

Ahlfeld, F.: Die Missbildungen des Menschen, Leipzig, F. W. Grunow, 1880-1882, vol. 1.

Arnheim: Ueber einen Fall von congenitaler halbseitiger Hypertrophie mit angeborenen Bronckiektasien, Virchows Arch. f. path. Anat. **154**:300, 1898.

Ballance, C. A.: Hemihypertrophy of the Head and Hypertrophy of Certain Toes, Trans. Med. Soc. Lond. **13**:516, 1889-1890.

Ballantyne, J. W.: Manual of Antenatal Pathology and Hygiene, Edinburgh, W. Greene & Sons, 1904, p. 257.

Bankhart, A. S. B.: A Case of Hypertrophy of the Right Side of the Face, Proc. Roy. Soc. Med., Lond. **9**: Sect. Stud. Dis. Child., 77, 1915-1916.

Bassoe, P.: Unilateral Hypertrophy Involving the Entire Left Side of the Body, Worcester State Hosp. Papers **61**:1912-1913.

Bateson, W.: Problems of Genetics, New Haven, Yale Univ. Press, 1913, p. 258.

Beck: Abnorme Grösse des Daumens und des Zeigefingers der rechten Hand, Med. Ann. von Chelius u. Naegle, Heidelberg **2**:89, 1836.

Bornaud: Observations histologiques sur un cas d'hypertrophie musculaires unilateral, Lausanne, 1891.

Broca: Journal of Physiology, Brown-Séquard **2**:70, 1859.

Brüning, H.: Ueber angeborenen, halbseitigen Riesenwuchs. München. med. Wchnschr. **51**:385, 1904.

Bull, G. J.: A Case of Bifurcated Foot with Eleven Toes, Boston M. & S. J. **93**:293, 1875.

Burlet: Gaz. méd. dé Lyon, July, 1862, p. 309.

Cagiati, L.: Klinschr. und pathologischer Beitrag zum Studien der halbseitigen Hypertrophie, Deutsch. Ztschr. f. Nervenheilk. **32**:282, 1907.

Chassaignac: Hypertrophie congénitale des deux membres droits, Bull. Soc. de chir. de Paris **8**:452, 1857-1858.

Cohen, Hyman: A Case of Hemihypertrophy with Increased Sugar Tolerance, J. A. M. A. **69**:463 (Aug. 11) 1917.

Curling, T. B.: Case of Remarkable Hypertrophy of the Fingers in a Girl, Med. Chir. Trans. Lond. **28**:452, 1845.

Davidson, A. J.: A Case of True Congenital Unilateral Hypertrophy, Med. Rec. **80**:420, 1911.

Demme: Ueber einen eigenthümlichen Fall von angeborener halbseitiger muskel Hypertrophie, Wien. med. Bl. **13**:17, 1891.

Desvouges: Bull. Soc. anat. de Paris **31**:510, 1855.

Eaton: Brit. M. J. **1**: 1886.

Fatichi: Sperimentale, Fir., 1895.

Finlayson, J.: On the Case of a Child Affected with Congenital Unilateral Hypertrophy and Patches of Cutaneous Congestion, Glasgow M. J. Ser. 4 **22**:327, 1884.

Fortescue-Brickdale, J. M.: A Case of Congenital Hemihypertrophy, Lancet **189**:10, 1915.

Foucher: Excessive Development of the Left Arm and Leg, Bull. Soc. anat. de Paris **25**:108, 1850.

Friedberg, H.: Riesenwuchs des rechten Beines, Virchows Arch. f. path. Anat. **40**:353, 1867.

Friedreich, N.: Ueber congenitale halseitige Kopfhypertrophie, Virchows Arch. f. path. Anat. **28**:474, 1863.

Gesell, A.: Present article. See also Psychol. Bull. (Summary) January, 1919.

Goddard, H. H.: The Causes and Consequences of Feeblemindedness, New York, Macmillan Co., 1914.

Goddard, H. H.: The Height and Weight of Feebleminded Children, J. Nerv. & Ment. Dis. **39**:217, 1912.

Gordinier, H. C.: A Case of Unilateral Hypertrophy of the Whole Left Side with Necropsy, Albany M. Ann. **39**:47, 1918.

Gould: Anomalies and Curiosities of Medicine.

Greig, D. D.: Unilateral Hypertrophy, Edinburgh Hosp. Repts. **5**:212, 1898.

Gruber, W.: Ueber einen Fall von Macrodactylie bei einen Lebenden, Virchows Arch. f. path. Anat. **56**:416, 1872.

Harrison, R. G.: On Relations of Symmetry in Transplanted Limbs, J. Exper. Zool., Balt. **32**:1, 1921.

Hoffmann, M.: Zur Pathologie des angeborenen, partiellen Riesenwuchses, Beitr. z. klin. Chir. Tübingen **48**:391, 1906.

Hornstein, S.: Ein Fall von halbseitigen Riesenwuchs, Virchows Arch. f. path. Anat. **133**:440, 1893.

Hutchinson, J.: Hypertrophy of the Left Side of the Body in an Infant, Rept. Soc. Stud. Dis. Child., Lond. **3**:194, 1902.

Hutchinson, R.: Report of a Case of Hemi-Hypertrophy with Post Mortem Examination, Proc. Roy. Soc. Med., Lond. **9**: Sect. Stud. Dis. Child. 66, 1915-1916.

Jacobi, A.: Am. J. Obst. **4**:321, 1871.

Lapage, C. P.: Feeblemindedness in Children of School Age, Manchester, Univ. Press, 1911.

Langlet: Hypertrophie congénitale de la moitié droite du corps, Union méd. et scient. du nord-est, Reims **6**:276, 1882.

Leblanc, E.: Thése pour le doctorat en médecine. Contribution a l'etude de l'hypertrophie congénitale, unilaterale partielle ou complete, Paris, Henri Jouve, 1897.

Lewin, G.: Studien über die bei halbseitigen Atrophien und Hypertrophien, namentlich des Gesichtes, Charité-Ann., Berl. **9**:619, 1884.

Logan: Hypertrophy of the Right Half of the Body in a Child Aged Four Years, New Orleans J. M. **21**:733, 1868.

McConnell, J. W.: A Case of Hemihypertrophy, Arch. Neurol. & Psychiat. **2**:136 (July) 1919.

McConnell, J. W.: Hemihypertrophy, J. Nerv. & Ment. Dis. **50**:348, 1919.

McGregor, A. N.: A Remarkable Case of Unilateral Hypertrophy in a Child, Glasgow M. J. **41**:189, 1894.

Maschke, A. S.: Unilateral Hypertrophy of the Leg with Congenital Misplacement of Striped Muscle Tissue of the Foot, Cleveland M. J. **6**:194, 1907.

Milne, B.: Report of a Case of Congenital Asymmetry, Lancet **1**:752, 1895.

Milne, J. B.: On the Case of a Child Affected with Congenital Symmetry of the Two Halves of the Body, Quart. M. J., Sheffield **3**:245, 1894-1895.

Möbius: Ueber Hemihypertrophy, München. med. Wchnschr. **37**:751, 1890.

Monod, A., and Trélat, U.: De l'hypertrophie unilatérale partielle ou totale du corps, Arch. gén. de méd. **1**:536; 676, 1869

Morgan, T. H., and Bridges, C. B.: Contributions to the Genetics of Drosophilia Melangaster (The Origin of Gynandromorphs) Washington, Carnegie Institute, 1919.

Mummery, P. L.: A Case of Hemihypertrophy, Proc. Roy. Soc. Med., Lond. **1**: Clin. Sect. 61, 1907-1908.

Newman, H. H.: The Biology of Twins, Chicago, Univ. of Chicago Press, 1917.

Newman, H. H.: Heredity and Organic Symmetry in Armadillo Quadruplets. II. Mode or Inheritance of Double Scutes and a Discussion of Organic Symmetry, Biol. Bull. **30**:173, 1916.

Ollier: Gaz. méd. de Lyon, 1862.

Osler, W.: Case of Congenital and Progressive Hypertrophy of the Right Upper Extremity, J. Anat. & Physiol., Lond. **14**:10, 1879-1880.

Parhon, C. J., and Severin: Sur un cas d'hémi-hypertrophie cranio-facio-linguale, avec trophoedème facial, N. iconog. de la Saltpêtrière **28**:282, 1916-1917.

Passauer, O.: Angeborne Hyperplasie der linken Gesichtshälfte, Virchows Arch. f. path. Anat. **37**:410, 1866.

Pershing, H. T.: Unilateral Hypertrophy of Limbs; Report of a Case, J. Nerv. & Ment. Dis. **40**:46 also 65, 1915.

Pollosson: Lyon méd., 1884.

Redard, P.: De l'hypertrophie congénitale partielle, Arch. gén. de méd. **1**:31, 1890.

Reismann: Un cas de gigantisme unilateral avec hypertrophie de l'hemisphere cerebrale du cete opposé, Ann. de méd. et chir. **6**:793, 1902.

Reismann: Further Note on a Case of Unilateral Gigantism, Australasian M. Gaz. **23**:280, 1904.

Schiller, M.: Totale Hypertrophie einer Körperhälfte, Berl. klin. Wchnschr. **47**:366, 1910.

Silverstrini, L.: Su di un caso d'ipertrofia congenita parziale, Clin. chir. Milano, **22**:581, 1914.

Speese, J.: Unilateral congenital hypertrophy, Arch. Pediat. **31**:278, 1914.

Steffen, W.: Angeborene Hypertrophie der einen Körpeherhälfte, Jahrb. f. Kinderh. **38**:379, 1894.

Telford, E. D.: Hemi-Hypertrophy of the Body with Naevus and Varicose veins, Lancet **2**:129, 1912.

Thomson, J.: On a Case of Myxoedematoid Swelling of One Half of the Body in a Sporadic Cretin, Edinburgh M. J. **37**:249, 1891.

Tilanus, C. B.: Ueber einen Fall von hemihypertrophia dextra, München. med. Wchnschr. **40**:65, 1893.

Tuckerman, F.: Some Observations in Reference to Bilateral Asymmetry of Form and Function, J. Anat. & Physiol. **19**:307, 1885.

Turbil: Gior. d. r. Accad. di med. di Torino, 1896.

Velebil, A.: Hemihypertrofia congenitalis, Rev. v neuropsychopath., Praha **10**:577, 1913.

Wagner, P.: Zur Casuistik des angeborenen und erwobenen Riesenwuchses, Ztschr. f. Chir., Leipzig, **26**:281, 1887.

Widenmann, A.: Ueber partiellen Riesenwuchs. Bietr. z. klin. Chir., Tübingen, **8**:625, 1892.

Wieland, E.: Zur Pathologie der dystrophischen Form des angeborenen partiellen Riesenwuchses, Jahrb. f. Kinderh., Liepz. **65**:519, 1907.

Wittelshöfer, R.: Ueber angeborenen Riesenwuchs der oberen und unteren Extremitäten, Arch. f. klin. Chir. **24**:57, 1879.

EYE SIGNS IN INTRACRANIAL TUMORS OF THE ANTERIOR FOSSA

WITH REPORT OF TWO CASES*

ALBERT B. SIEWERS, M.D.
SYRACUSE

Retrobulbar neuritis with central scotoma and primary optic atrophy on the side of the lesion and concomitant papilledema in the opposite eye as a syndrome in intracranial tumors of the anterior fossa was stressed by Kennedy[1] in 1911. In the same year Paton and Holmes, in a study of brain tumors, state:

> We have examined a case in which a basal tumor compressed one optic nerve before its exit through the optic foramen and produced a retrograde atrophy of the nerve fibers and ganglion cells, and sclerosis of the nerve and disc. There was no edema . . . In the other eye there was typical papilledema.

Similar cases had been observed in isolated instances. Willbrand and Saenger[2] quote Seydel, who observed in a man aged 47, slight paresis of the right half of the body and trembling of the extremities, beginning in the right arm. At the age of 32, he had received a blow on the head followed by unconsciousness for several weeks. Ophthalmoscopic examination revealed papilledema in the right eye and in the left a pale nerve head. There was an oval shaped absolute central scotoma on the left. Removal of an extradural fibroma in the anterior fossa brought about regression of symptoms.

The same authors cite two other cases—one of frontal tumor on the under surface, more on the left than on the right. The left optic nerve was directly compressed. The result was primary atrophy of the left and intense neuritis of the right optic nerve. The other was a case of right frontal tumor. The right papilla appeared normal, but the patient was blind in this eye. The left disk showed a swelling of 2 diopters. Visual acuity was good on the left.

REPORT OF CASES

Case 1.—*History.*—About Sept. 1, 1920, C. J., a fire department chauffeur, 38 years old, began to complain of pain in the right occipital region; it recurred spasmodically. Soon after, he began to have attacks of uncontrollable coarse shaking of the right arm. He was several times incontinent of urine while going about his business and incontinent of feces once while in bed. He was also impotent. By the middle of October he had to give up work.

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*From the psychiatric and Surgical Service of the Johns Hopkins Hospital.

[1] Kennedy, Foster: Am. J. Med. Sc. **142**:355 (Sept.) 1911; J. A. M. A. **67**: 1361 (Nov. 4) 1916.

[2] Willbrand and Saenger: Centralbl. Chir. 1896, No. 3.

After December 1, he vomited several times without nausea and with considerable force. During the week preceding his admission he had difficulty in walking, and seemed rather dull. On December 20, he came to the Henry Phipps Psychiatric Clinic with this dulness, occipital headache, and attacks of trembling of the right arm. He was admitted December 23.

He was correctly oriented and showed slight "immediate memory" defect; that is, his retention was only four digits, probably accounted for by poor attention and poor cooperation. At times he was confused and restless; he would get in and out of bed repeatedly. He said: "I feel fine except when I have that pain."

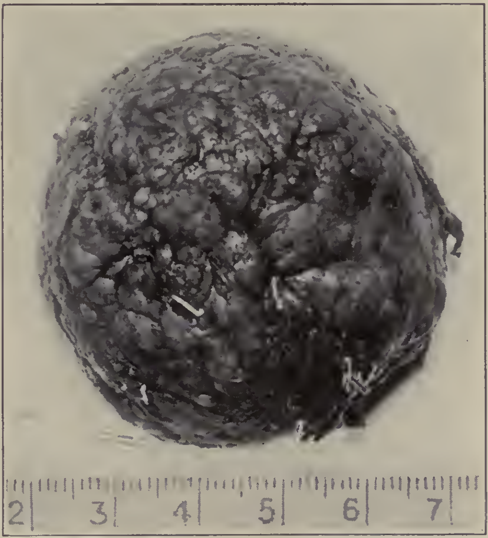

Fig. 1.—(Case 1).—Tumor on under surface of frontal lobe.

Examination.—The patient lay in bed with eyes and head turned toward the right. He tended constantly to roll out of bed on the right side. He could smell camphor and peppermint, but could not name them or tell their nature. (The left eye had been blind for some time and had been more prominent than the right since the beginning of the illness.) The left disk was atrophied. There was beginning papilledema in the right eye. The pupils were regular, the right slightly smaller. The right reacted well to light and accommodation, but the left did not react. There was a coarse, rhythmic tremor of the right arm; rate, four to five per second. The finger-nose test showed some ataxia and there was dysdiadochokinesis of both hands. Abdominal wall reflexes were absent. The cremasteric was active on both sides. The deep reflexes were normal and the plantar response flexor on both sides. The gait was reeling, the patient falling backward and to the right. He could not stand unsupported.

During the examination the patient appeared to have several petit mal attacks, of which he had no realization. No disorder of the internal organs was found.

The Wassermann reaction on the blood and spinal fluid was negative. The fluid showed increased globulin and 3 cells. The roentgen-ray picture of the skull was negative.

Because of the headache and vomiting, repeated incontinence and dulness, protrusion of the left eye, atrophy of the left and edema of the right nerve-head, tendency to deviation to the right, the tremor attacks of the right arm and postural difficulty, a diagnosis of left frontal tumor was made, and the patient was transferred to the department of surgery.

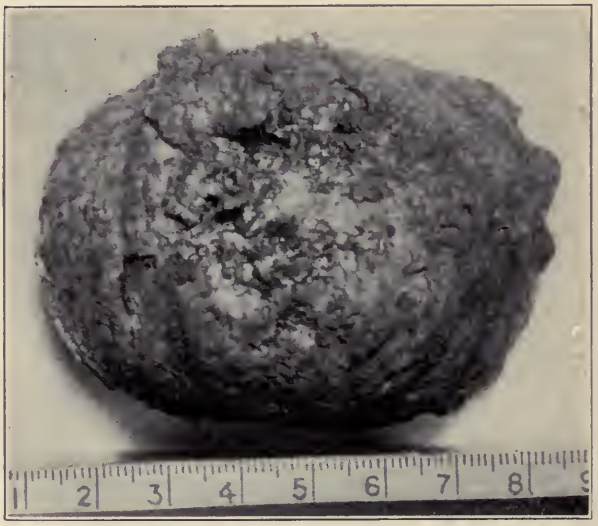

Fig. 2.—(Case 2).—Tumor, arising from dura, located under right frontal lobe.

Treatment and Course.—On December 29, Dr. W. E. Dandy found that only the right lateral ventricle filled with air. Two days later he exposed the left frontal lobe and found a diffuse dural endothelioma with a large mass of tumor on the under surface of the frontal lobe. The tumor could not be entirely removed. Figure 1 shows the size of the removed part.

The patient made an uneventful operative recovery and after two weeks was able to go home. Later he received several radium treatments. On June 1, 1921, he reported feeling perfectly well and was back at work in the fire department, but not driving a car.

Laboratory Findings.—The dural surface has the same appearance as that part of which spread diffusely over the under surface of the dura. There was a "crust or frosting" of tumor half an inch thick, adherent to the dura. It extended to the longitudinal sinus above, as far back over the occipital lobe as one could see, down for a short distance over the temporal lobe and nearly to the midline in front.

CASE 2.—*History.*—W. S., aged 51, a laborer, formerly an electrician, was admitted to the Johns Hopkins Hospital, on Aug. 6, 1920, complaining of "spells" accompanied by an odor of sewer gas. Eight years ago he received an electric shock from an 18,000 volt circuit. Some "black blood" discharged from his nose a week later. He continued at work but "nervousness" not complained of previously now caused changes in occupation more or less frequently.

In May, 1918, he suddenly became "light-headed" and noticed a strong smell of sewer gas. He was working on a pipe line at the time and paid no attention to the occurrence. Attacks similar to this then came on two or three times daily and lasted two or three minutes. In the attacks there were color hallucinations. Any object that he happened to be looking at seemed to become red or green as if looked at with a colored glass. These hallucinations gradually disappeared.

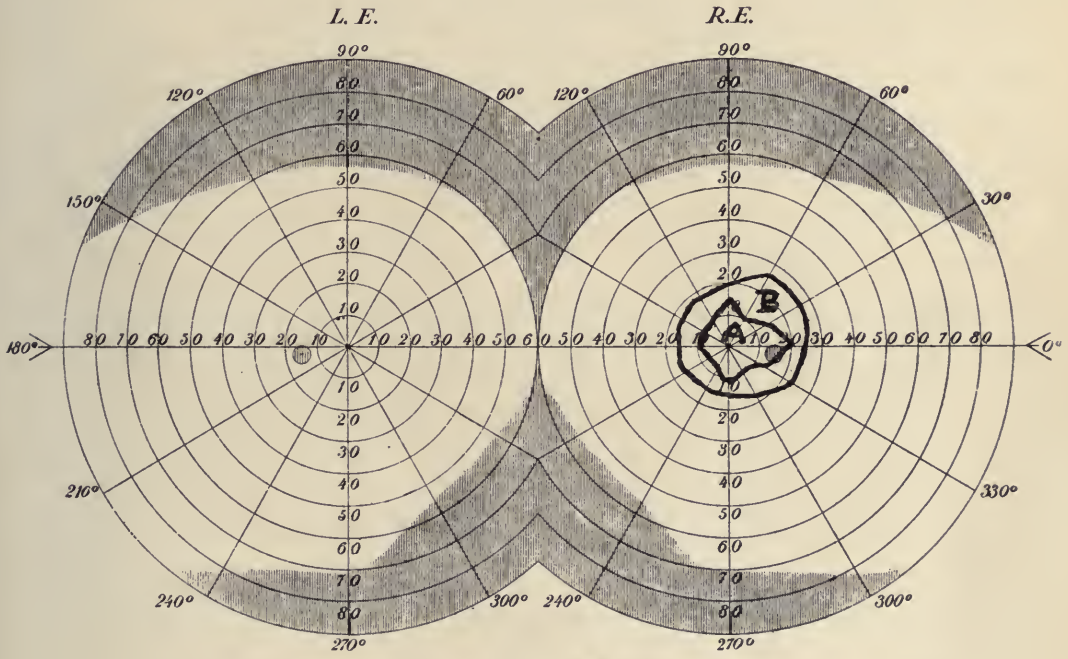

Fig. 3.—(Case 2).—Fields of vision; *A*, absolute scotoma; *B*, a larger bordering scotoma for red and green.

The visual fields of C. J. (Case 1) were not charted. He was totally blind in the left eye. The right visual field was normal.

In February, 1919, he had an attack that started the same way but passed into a generalized convulsion with loss of consciousness. He had had five such attacks.

Since July, 1919, he had complained of headache and of inability to see with the right eye an object directly in front of him. He had become more irritable, had lost 35 pounds in weight, and for the last year had had no sexual desire or power. (It will be noted that the patient in Case 1 had also lost his sexual power.)

Examination.—The patient recognized test odors. The pupils were normal. Vision was good except for a central scotoma on the right. The right disk was beginning to atrophy, especially on the temporal side. There was beginning papilledema on the left. The abdominal reflexes were present; the left was reduced and easily exhausted. The cremasteric reflexes were active and equal and the deep reflexes normal. The plantar response was flexor. There was dysdiadochokinesia of the right hand.

The internal organs were normal. The blood and spinal fluid Wassermann reactions were negative. The fluid showed an increase in globulin. Roentgen-ray examination revealed increased density over the right frontal region.

Treatment and Course.—A diagnosis of right frontal neoplasm was made, and on Aug. 11, 1920, Dr. W. E. Dandy exposed the right frontal lobe. A tumor arising from the dura under the right frontal lobe and extending backward was enucleated. It was very vascular and weighed 62 gm. There was terrific bleeding; the patient was shocked and died the following evening.

COMMENT

It will be noted in the two cases that the eye signs as described by Kennedy and others were very prominent and of considerable significance in the diagnosis of the condition and of the localization of the lesion.[3]

405 Fayette Park.

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[3] In addition to the references given the following may be of interest: Paton, Leslie: Brain **32**:64-91, 1909-1910; Paton and Holmes: Ibid. **33**:420, 1916-1917; Willbrand and Saenger: Die Neurologie des Auges, **2**:535 and 586.

RESULTS IN TWO HUNDRED AND FIFTY-ONE CASES FIVE YEARS AFTER ADMISSION TO A HOSPITAL FOR MENTAL DISEASES

EARL D. BOND, M.D.

PHILADELPHIA

A study has been made of what happened to two hundred and fifty-one consecutive women patients in the five years following their admission to the department for mental and nervous diseases of the Pennsylvania Hospital. We have records of isolated cases that have been followed for a life-time, but they lack the authority that comes from unselected consecutive cases. The fact that this as a private hospital has insisted on having responsible families and friends stand behind its patients has made possible this laborious undertaking.

The charts and tables show the condition of each of the 251 patients through five years. In the charts an individual can be followed by reading down a numbered column; psychosis unimproved is shown by close hatched lines, improved (usually fully able to look after herself at home) by lines more widely spaced, and recovery is shown by white spaces closed in at the bottom by a line indicating the time of the last report. More concisely, the more cross lines the more illness.

The list is continuous, but for convenience in reference has been broken into four parts—A, B, C, D.

RECOVERIES

In Group A Patient 1 is shown in the chart as sick for three and a half months, then as making a good recovery which she has consistently held for nearly five years. At the end of the fourth year after admission she was found by the follow-up nurse at her former home and appeared well. At the end of the fifth year when the nurse arrived she was out marketing, but her mother and the neighbors said that she was in very good health. She was a married woman, aged 30, who had had one child before admission and none since. Other cases, up to Patient 30, follow the same paradigm; the illness has varied within narrow limits, except in Patient 8. And yet, the obstinacy of the attack in this instance, over three years in a patient aged 53 with a hemiplegia, has given way to recovery which has restored a woman of fine character and high ability to a world which needed her. On leaving the hospital well she went to a distant state and saw her only son embark for France. She awoke one morning to find her husband lying beside her bed unconscious from the same kind of cerebral hemorrhage from which she once suffered and nursed him through a long and hard illness to his death, after which she broke up her home and has acted as administratrix of the estate. Patient 30 herself, a woman of 60, made a good recovery and died at home over two years later of "cardiac asthma" without any mental symptoms. Patients 31, 32 and 33 had long drawn out cases of involutional melancholia with good endings; all reached the point of emaciation; Patient 33 remained at this hospital during the whole psychosis; Patient 31 recovered at the Norristown State Hospital; Patient 32 tried three other hospitals and recovered under the care of a nurse at home. She is "as well as ever in her life, but much more amiable." Patient 34 after two short attacks and some symptoms which showed after she went home has been very well for over two years and is holding a much better position than her abilities before the first attack seemed to warrant.

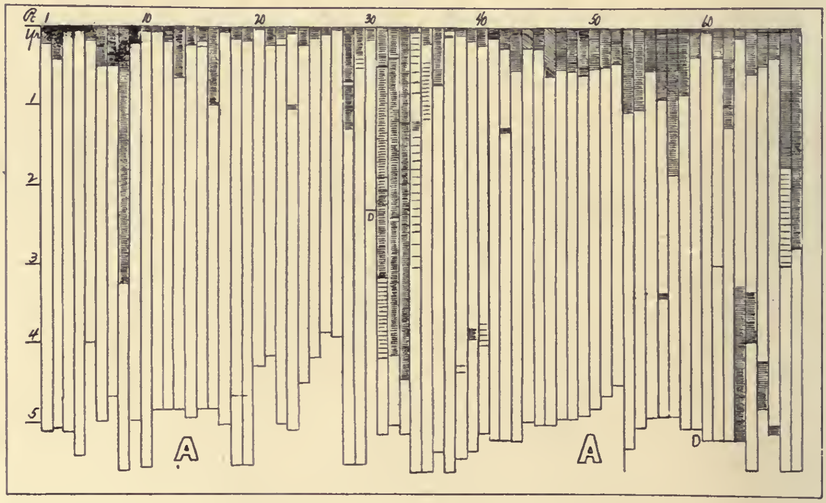

Chart 1.—Review of five year period.

The amazing and cheering thing in these first sixty-eight cases is the spectacle of normal life regained—even for a five-year period. The total is over two centuries and a half. Sixty patients have met the test of "sustained self-adjustment to life outside" and do not at present show signs of breaking down. The facts found about these sixty-eight out of 251 patients show well enough what "recovery" can mean and whether or not it is worth while.

Table 1 gives a list of the sixty-eight patients in the same order as on the chart, including the age, the diagnosis (as of present date) and features which are unusual. As these studies bear on the social importance of a recovery, an effort has been made to find the number of children born since the last admission, and these are noted with the number born earlier.

A summary of diagnoses for this group of sixty-eight maintained recoveries shows that there were 40 manic-depressive psychoses cases, 14 unclassed, 6 involution melancholias, with an average hospital treatment of over two years, 3 psychoses with infections, 3 psychoneuroses, 1 psychosis with morphia and 1 dementia praecox.

These patients had had sixty-five children before the attack for which they were admitted, four of whom had died. Since the attack only two children had been born to them, according to our information, which is quite accurate. Three patients, after waiting for four or five years after recovery, were married.

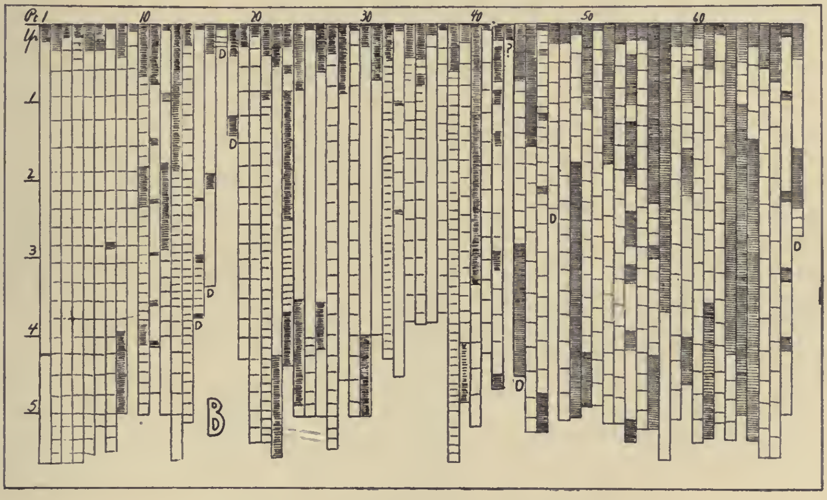

Chart 2.—Review of five year period.

PARTIAL RECOVERIES

Cases collected under B show as striking a tendency to improve as do those under A, but are as strikingly differentiated by coming to a partial or poorly maintained adjustment. In the sixty-nine cases here grouped the improvement is manifested as early as in the sixty-eight of the first group, and all but five have become capable of self-support.

Patient 1 was sick three months and for five years since has been at home, capably managing a household of four people without help, but "nervous." Four years after recovery she had a severe cold accompanied by headache and delirium for two weeks.

TABLE 1.—Age, Diagnosis and Unusual Features in Cases of Sixty-Eight Patients Recovered

No.	Age	Diagnosis	Comment
A1	30	Manic depressive	One child before, none since
A2	40	Manic depressive	One child and one induced miscarriage before, none since; back fractured by leaping out of window; good physical result
A3	62	Manic depressive	Single
A4	58	Manic depressive	Three children before
A5	26	Manic depressive	Married four years after admission
A6	49	Manic depressive	Two children before, none since
A7	25	Manic depressive	Single
A8	53	Manic depressive	Hemiplegia; one child before, none since (see text)
A9	26	Psychosis with infection	One child at beginning of this attack, none since
A10	35	Manic depressive	Three children before, 1 died, none since
A11	30	Manic depressive	Single
A12	25	Unclassed	Married at end of fifth year of recovery
A13	26	Manic depressive	No children
A14	55	Manic depressive	Four children before attack of whom three died; living child gives Wassermann +
A15	38	Unclassed	Divorced; no children
A16	35	Manic depressive (infection)	One child before attack
A17	46	Manic depressive	Single
A18	28	Unclassed	Divorced, no children
A19	29	Dementia praecox	Single; two years before admission her father died; months before admission her minister died; the patient showed unusual grief. For five months before admission and two months in the hospital she was reticent, uninterested in her surroundings; expressed somatic delusions (stoniness and iciness); thought that she made other people sick; speculated on telepathy, on how she could hold "the dream life." She had been fond of her father, but said she had no real affection for her mother; recovery after a little over two months in hospital; has been carefully followed since; she has entered and graduated from college and is holding a responsible position. She still thinks of her illness as a "fight between the real and the unreal"
A20	19	Psychosis with infection	Single
A21	50	Unclassed	One child before attack none since
A22	43	Involutional melancholia	No children
A23	59	Involutional melancholia	One child; menopause at 43
A24	43	Involutional melancholia	Single
A25	58	Manic depressive	No children
A26	24	Unclassed	One child before
A27	47	Manic depressive	Married, has had no children
A28	34	Manic depressive	Four children before attack, none since. This patient made first a recovery which lasted only a month, but after a relapse of six months has remained well four years and is now a competent housekeeper for her family of five, and carries on and enjoys her social duties
A29	19	Manic depressive	A deaf mute; has shown for five years all the ability and poise which she had before the attack. As the deafmutism can be considered separately from her mental disturbance it seemed fair to mark her "recovered." Patients with a mental upset on a psychopathic inferior basis have been called "improved" when the psychosis cleared up
A30	66	Unclassed	Four children; died of asthma long after recovery
A31	48	Involutional melancholia	A widow with two children
A32	43	Involutional melancholia	Married; three children before, none since; in recovering increased from weight of 70 to 125 pounds
A33	50	Involutional melancholia	Married, one child before, none since
A34	19	Psychoneurosis	Single; a recovery with a gain of efficiency and poise over previous condition
A35	17	Unclassed	She was married five years and nine months after admission
A36	44	Morphia hallucinosis	Single; her brother reports that she has kept house without difficulty and has not to anyone's knowledge returned to her drug
A37	20	Unclassed	Married a year after recovery; deserted by her husband after the birth of one child; she is working as a saleswoman and supporting her child, but she is "still rather a rattlebrain"
A38	50	Manic depressive	Single; a depression which apparently replaced convulsions, and was filled with desperate suicidal attempts. She has been well, fat, and has held a good job for five years; in the summer of her fourth year she was tired and weak
A39	17	Manic depressive	Single; two sharp attacks of excitement with good recovery

TABLE 1.—Age, Diagnosis and Unusual Features in Cases of Sixty-Eight Patients—(*Continued*)

No.	Age	Diagnosis	Comment
A40	19	Manic depressive	Single; very well since early recovery with the exception of a mild melancholia in the fourth year; always well employed
A41	19	Psychoneurosis	Single; hysteria
A42	51	Manic depressive	Single
A43	12	Unclassed	Hallucinated or suicidal; now doing well in high school
A44	23	Unclassed	One child * (p. 393)
A45	54	Manic depressive	Widow; one son
A46	51	Manic depressive	Widow; no children
A47	21	Unclassed	Single * (p. 394)
A48	47	Manic depressive	Two children before
A49	34	Manic depressive	One child
A50	26	Manic depressive	One child; after the chart was drawn, the patient had another attack which is not indicated
A51	44	Manic depressive infection	Single
A52	58	Manic depressive	Seven children. She has had another attack since the chart was made; for several years she has been very deaf and sensitive; otherwise she has all her life seemed to be a hard working, well balanced individual
A53	43	Manic depressive	Seven children
A54	31	Manic depressive	One child
A55	23	Manic depressive	Single
A56	22	Manic depressive	Married; no children
A57	24	Manic depressive	Married; one child
A58	27	Manic depressive	Single; in better health and earning much more than before the attack
A59	45	Manic depressive	Single; died five years after recovery, of pneumonia, without any recurrence of mental symptoms
A60	35	Unclassed	Married; no children
A61	23	Manic depressive	One child; this patient was followed three years, during which time she actively assisted her husband in his store at Atlantic City; they moved suddenly and we have not been able to locate them
A62	34	Unclassed	Sent from here and from a state hospital unimproved; recovered when she heard that her husband was unfaithful* (p. 393); two children before admission, none since
A63	45	Manic depressive	Three children. The new attack was associated with virulent mouth infections
A64	39	Unclassed	Single
A65	21	Manic depressive	Single
A66	47	Manic depressive	Single
A67	24	Psychosis with infections	One child
A68	20	Psychosis with infections	One child before admission, one child since

* Bond, Earl D.: A Review of the Five-Year Period Following Admission in One Hundred and Eleven Mental Patients, Am. J. Insan. **76**: 385-394 (Jan.) 1921.

The cases of patients 2 to 8 illustrate the clearing from psychoses with residual symptoms, which either were produced by the psychosis or were part of an evident congenital or earlier acquired inferiority. Patient 2, for instance, backward, with many physical signs of an old anterior poliomyelitis and irritable, at the age of 25 became depressed and deluded; she recovered from this acute mental attack in 4 months and has been at home 5 years since in a cheerful, clear, but always partially dependent condition. Patient 3, sexually delinquent, at 18 had a hallucinosis, from which she recovered in two months; for five years since she has been self-supporting, and, according to the society which has followed up her case, she has been doing extremely well. Patients 3 and 4 made prompt recoveries from acute psychoses, but were left with a lack of insight (3) and eccentricities (4), which prevent our regarding them as well rounded recoveries. Patient 7 is probably the most efficient worker of the 251 patients, and her efficiency has increased since her mental attacks; she visits the hospital regularly and is nervous and restless. Patients 10, 15, 33, 42 and 44 show alternating phases of manic-depressive psychosis; all of them could be called recovered at many given points, and could swell a recovery rate. Patients 13 and 40 are recovering after long illnesses. Patient 25, an able business woman, has kept in close touch with the hospital since her recovery from a severe delirium at childbirth; while she has all her former ability, she has in the last year had attacks of petit mal.

This group of seventy-one patients (B) as a whole seems efficient but queer, while Group A seems well balanced. No new therapeutic procedure could be expected to attain much better results with Group A, but in Group B is much valuable material to be salvaged. Those patients who have gone quickly from one phase of manic-depressive psychosis to another have made the most unsatisfactory social record because of disturbances at each commitment and because two have made unwise marriages and each has given birth to a child.

This group of sixty-nine patients who showed a tendency to improve to the condition of self-support gives diagnoses of manic-depressive cases in 27 instances, involutional melancholia in 5 and dementia praecox in 9 cases, 15 remaining unclassed; 8 psychoses in psychopathic inferiors, 2 psychoneuroses, 2 psychoses with somatic disease and 1 alcoholic psychosis.

These patients had seventy-three children before they were admitted, of whom eight had died, one by suicide, and two have become state charges (insanity and desertion). Since admission they have had seven children, though by their very inclusion in this group the mothers are in no condition to look after them.

UNIMPROVED UNTIL DEATH

In Group C are included the fifty-one patients that showed no improvement and died, twenty-one of the deaths occurring away from this hospital. This group is not charted; 2 deaths occurred at over 90 years of age, 16 deaths between 90 and 60, 22 between 60 and 40 and 10 below 40.

Of the thirty deaths in hospital, nineteen resulted from recognized and chronic progressive diseases. Six others were covered by necropsy; three of these were described in a previous paper * and two in Dr. Strecker's † paper on "Late Catatonia." The other showed marked degeneration of the liver, kidney and cord. Three other patients died within eight days of infections existing before admission. The two remaining deaths were due to exophthalmic goiter and lobar pneumonia, but were favored by the exhaustion due to psychoses.

TABLE 2.—FINDINGS IN GROUP B IMPROVED

No.	Age	Diagnosis	Comment
B1	38	Unclassed	Three children before, none since
B2	25	Psychosis in inferior	No children
B3	18	Psychosis in inferior	Sex delinquent; no children
B4	26	Dementia praecox	No children
B5	57	Manic depressive	One child before (suicide); none since
B6	44	Manic depressive	No children
B7	34	Unclassed	Three children before; none since
B8	48	Dementia praecox	No children
B9	31	Psychosis in inferior	Married since; (?) children
B10	37	Manic depressive	No children
B11	49	Manic depressive	Two children; none since
B12	38	Manic depressive	Four children before, 1 child since
B13	42	Involutional melancholia	No children
B14	34	Manic depressive	One child before, none since
B15	51	Manic depressive	Eleventh attack; death in fourteenth attack; single
B16	27	Manic depressive	Fever (105 F.) with congenital aortic stenosis; recovery from this attack and another two years later; death at home after three years of pneumonia. Married; no children
B17	42	Involutional melancholia	Some improvement before death of uterine cancer; widow; one son in state hospital; dementia praecox
B18	19	Unclassed	Hyperthyroidism; death in second attack in state hospital; single
B19	45	Dementia praecox	Improved enough to go home where she did poorly; under osteopathy for three months, then fairly well, but very peculiar; three children before, (?) since
B20	16	Psychopathically inferior	Sex offender, sterile; deaf; has been self-supporting under constant supervision
B21	40	Manic depressive	Twelfth attack; seven children before
B22	30	Manic depressive	Second attack; one child born before her admission here; now under care of Children's Aid; husband alcoholic
B23	28	Manic depressive	Married; no pregnancies
B24	41	Manic depressive	Married; four children
B25	36	Psychosis with somatic disease	One child, none since; described in text
B26	25	Unclassed	Single
B27	57	Alcoholic psychosis	Single
B28	30	Unclassed	Eccentric, but causes no trouble at home
B29	32	Unclassed	Single on admission but three years later married and had one child, husband died shortly after; patient is nervous but well; supports herself
B30	29	Manic depressive	Second attack; first attack was postpuerperal; has had three children; since admission one has died and another has been born
B31	57	Involutional melancholia	Single
B32	48	Involutional melancholia	Married; eight children, four of whom are dead; menopause at 46
B33	18	Manic depressive	Third attack; married three years after admission and has a baby
B34	21	Unclassed	Single; well employed
B35	23	Dementia praecox	Married; one child
B36	33	Psychosis with constitutional inferiority	Fourth attack; had two children (one illegitimate) before admission; divorced
B37	50	Paranoid condition	Single; queer and nervous but a good worker; more than self supporting
B38	42	Unclassed	Married; two children before this attack, none since. While she has done the housekeeping for herself and children, she has constantly quarreled with her husband and twice sent him to jail
B39	23	Dementia praecox	An attack with a pseudorecovery; at home seclusive and without insight until an attack four years later, for which she is still under treatment; single
B40	38	Involutional melancholia	Single; a three-year attack with "recovery" marked by lack of insight, but ability to do her work at home
B41	52	Psychoneurosis	A widow with two children; while much better when she left the hospital and able to gain ten pounds, she is now a dispensary rounder
B42	26	Manic depressive	The circular type, barely getting an emotional balance for a week or so between attacks; in her excitements she has been much in the newspapers, has made an unfortunate marriage, and has had a baby
B43	20	Dementia praecox	Lost after discharge from another hospital; single
B44	29	Manic depressive	Married; no children; died in second attack
B45	40	Manic depressive	Single
B46	18	Dementia praecox	Single

TABLE 2.—FINDINGS IN GROUP B—(*Continued*)

No.	Age	Diagnosis	Comment
B47	67	Unclassed	Married; no children; died of heart failure after two years of good health at home
B48	41	Constitutional psychopath	Married; no children
B49	55	Unclassed	Single
B50	35	Manic depressive	Single; able to work as a private nurse for three years
B51	55	Manic depressive	Fifth attack; eleven daughters of whom one died; at home doing much good work for her church
B52	41	Psychosis with somatic disease	After birth of second child;† recovered after seventeen years' illness, went home and came back for a second admission; this time she gradually completed making adjustments to new conditions and was able to leave the hospital and remain outside
B53	58	Manic depressive	Single
B54	63	Manic depressive	Manic attacks; one child
B55	18	Constitutional psychopath	Single; no children
B56	45	Manic depressive	Circular; single
B57	20	Manic depressive	Long attacks; for the last fifteen months a pupil nurse in a New York hospital
B58	54	Manic depressive	Depression; three children
B59	45	Manic depressive	Circular; never keeping her balance between attacks; single
B60	25	Constitutional psychopath	Two children before admission of whom one has since died; now six months pregnant; hypochondriacal
B61	42	Dementia praecox	Married; one child
B62	47	Unclassed	Single
B63	40	Dementia praecox	Single; at home; eccentric
B64	42	Unclassed	Married; no children
B65	55	Manic depressive	Attacks coalesced; two children
B66	52	Manic depressive	Married; no children; has been nervous and seclusive, but able to teach in the high school
B67	28	Unclassed	Married; one child before admission and one since
B68	48	Psychoneurosis	Married; three children; very well and capable between attacks
B69	29	Unclassed	Single; two attacks with marked improvement followed by death of an unknown cause at home

† Strecker, E. A.: Certain of the Clinical Aspects of "Late Catatonia" with a Report of Cases, Am. J. Insan. **74**: 185-220 (Oct.) 1917. A Psychosis of Seventeen Years' Duration with Recovery, J. A. M. A. **64**: 1151-1154, 1915.

Of the twenty-one deaths outside of the hospital, the causes were plainly evident in eleven on discharge from the hospital. Two others had nephritis and high blood pressure and died after two years at home; the type of illness seemed to be manic-depressive. Six patients died at home and two in other hospitals, from one to four years after discharge.

It should be remembered that all of the fifty-one deaths accounted for occurred with no marked improvement in the patient before death. A few others showed a tendency to improve before death and are classified elsewhere. The ages given on the chart and the diagnoses in Table 3 show that there is not much to expect in this group from new therapy. In the younger patients it is a matter of earlier treatment, of preventing infection (abortion, acute nephritis, syphilis) and diabetes, pellagra, exophthalmic goiter and of recognizing brain tumor—chiefly before the patient reaches the hospital.

This group of fifty-one patients, who had fatal illnesses, had had seventy-one children, of whom seven had died; there is a definite history of ten abortions. The average age is, of course, much greater than that of any other group. Many children were adults before the onset of the mothers' illness. No children were born after admission to this hospital.

UNIMPROVED

Group D, consisting of sixty-three cases, presents a sad monotony. Fifty-two are still under hospital care; one patient with a drug psychosis kept constantly to her addiction and was committed to several hospitals until the end of the five-year period when the chart was made—since then we have learned of her death from an overdose of chloroform. Patients D 30 and 31 could be followed for only three and one-half and four years.

As there are practically no other differences in the careers of these unimproved patients, details will be given grouped by diagnosis instead of on a chart.

TABLE 3.—Diagnosis in Fifty-One Cases and Number of Deaths

Senile dementia	5
Psychosis with cerebral arteriosclerosis	8
Involutional melancholia	1
Involutional melancholia with tuberculosis	1
Involutional melancholia with diabetes	1
Multiple sclerosis	1
Psychosis with brain tumor	2
General paralysis (3); juvenile (1)	4
Psychosis with nephritis	1
Psychosis with nepritis, hepatitis, myelitis	1
Psychosis with pellagra	1
Psychosis with acute infection	8
Manic depressive	9
Dementia praecox	3
Unclassed	5
Total	51

Dementia praecox cases numbered thirty-four and all but three were in hospital after five years, three being under home care. The ages at admission were 18, 19, 20, 20, 25, 25, 25, 26, 27, 27, 28, 28, 28, 31, 32, 32, 33, 34, 35, 37, 37, 38, 38, 39, 43, 46, 46, 46, 47, 51, 54, 56 and 58. The ages tend to indicate late admission to the hospital, which is borne out by the histories. Naturally hospital residence has kept these patients from having children. Before admission they had had twenty-seven in all, one of whom was a musical prodigy, and another the apparent cause of the mother's chronic disease, the only living issue obtained by special precautions and caesarian section after three miscarriages.

Psychoses with senility and arteriosclerosis number four, all of them still under hospital care. The ages at admission were 71, 60, 72 and 62. There were no children.

Of manic-depressive patients, six remain essentially unimproved—three of mixed type with admission at the ages of 48, 50 and 52, one depressed admitted at 73 and two of manic type admitted at the ages of 43 and 58. None of these have had children since their admission as patients; they had had twenty before.

Two patients with epileptic dementia, admitted at the ages of 10 and 42, are now worse. The latter had had three children.

Four patients with drug psychoses progressed unfavorably, one of them taking an overdose, after the five-year period was charted. They were admitted at the ages of 30, 38, 38 and 46. Before admission one patient had had two children and two others, one each; they had none afterward. The fourth patient had no children and was divorced; it is not certain that she has had no children in the five-year period, but it is unlikely.

Three psychoses in psychopathic patients aged 29, 40 and 37 remain unchanged. There had been a record of one child (and several abortions) in this group.

One feebleminded girl is now in a home (aged 12).

TABLE 4.—Diagnosis of Two Hundred and Fifty-One Cases

Manic depressive	82
Dementia praecox	47
Unclassed	42
Senile-arteriosclerosis	17
Psychoses with somatic disease	17
Involutional melancholia	14
Psychopathic inferiority	11
Drug psychoses	6
Psychoneuroses	5
General paralysis	4
Organic brain disease	3
Epileptic psychoses	2
Feebleminded	1
Total	251

A patient whose psychosis seemed closely associated with heart disease, whose blood pressure at the age of 40 was systolic 230 and diastolic 104, is still living in the department for mental diseases of the Philadelphia Hospital (single).

The condition of eight unclassified patients was left unchanged. The ages at admission were 31, 38, 63, 28, 47, 58, 22 and 51. Fourteen children were born to them before admission.

In this entire group of unimproved, the patients had sixty-eight children.

SUMMARY

These 251 patients were admitted five years ago in the ordinary course of hospital work. They were consecutive admissions, received on the average some time after the onset, and divided by diagnoses as indicated in Table 4.

Of this big group, sixty-eight patients have gone back to the community fully recovered, sixty-one have gone back to the community on a self-supporting basis, eight have shown marked improvement, in fifty-one the illness has progressed to death, and in sixty-three there has been essentially no improvement. New therapy has its greatest opportunity in the second and fourth groups; it may stabilize those tottering patients who show a strong tendency to improve in any case, and anything it can do in the unfortunate last group will be clear and striking gain. The deaths may be lessened by gains rather in general medical knowledge than in psychiatry—by procedures against infections and for prolonging life. A point worth emphasis is that a new therapeutic procedure must show tremendous results to better the record made by the first group under the usual procedures of 1914.

TABLE 5.—Results in Patients with Several Contrasting Diseases

	Recovered	Recovered Minus	Died	Unimproved
Dementia praecox	1	9	3	34
Manic depressive	40	27	9	6
Involutional melancholia	6	5	3	..
Senile-arteriosclerosis	..	..	13	4
Unclassified	14	17	5	8

What happened to patients with several contrasting diseases is shown in Table 5. It speaks for itself. The dementia praecox patients stay; the only deaths occurred in three who were taken home. The senile-arteriosclerotic patients die, although we all know patients who seem to be exceptions. The manic-depressive death rate is not inconsiderable; deaths during excitement were always partly due to another disease. The unclassified, a group kept large in order to keep other groups pure, had an even distribution of results; most of them were unproductive, resistive patients with no signs of organic brain disease.

The total number of children born to these 251 patients was 286, of whom nineteen died, leaving 267, or over one to a patient, nine of whom were born since the admission of their mothers to this hospital.

Abstracts from Current Literature

GRASP REFLEX WITH BRAIN TUMOR. A CONTRIBUTION TO THE STUDY OF THE LOCALIZATION OF THE CENTER OF SEVERAL COMPLICATED REFLEX MOVEMENTS. W. Parrisius, Deutsch. Ztschr. Nervenheil. **67**:29, 1920.

Parrisius bases his observations on the following case: Frau K., 47 years of age, was first observed August, 1919. Her left arm became weaker, which annoyed her in agricultural work, and her gait more clumsy, owing to weakness in the left lower extremity. For some time she had been unable to let go of utensils which she grasped with her left hand. In September she fell twice in an attempt to get out of bed; following that she suffered with headaches.

The physical findings on admission to the clinic in October were: Strength in the left arm and leg was somewhat reduced; there was considerable spasm of the arm and leg musculature. On the left the periosteal and tendon reflexes in the arm and leg were increased, and there was a slight foot clonus; distinct Babinski and Oppenheim reactions were present. Only the upper abdominal reflexes were obtained (flaccid epigastrium, pregnancy). Perhaps the speech was somewhat indistinct. The left hand could be voluntarily closed into a fist only quite slowly, but if one lay in her hand an object such as a handle of a percussion hammer, or if one touched the surface of the hand quite gently, it closed immediately, while the thumb and index finger grasped quite energetically, like the claw of a crayfish. The patient was unable to open the tightly clenched fist.

Nine days later there was a slow, distinct improvement of the hemiplegic appearance; however, the left hand could not be closed voluntarily on command. At the slightest touch of the palm the phenomena described took place. The reaction seemed to be greatest if the forearm was previously supinated. Then simultaneously with the closure of the fist pronation occurred. If one pulled on a seized object, the fist closed more firmly. If the patient accidentally came in contact with the slightest object, then closure of the fist occurred as a reflex action. A sister frequently had to loosen the fingers from an object. Abduction, adduction, pronation and supination movements in the wrist were awkward, and combined purposeful movements, such as grasping the nose or ear, and other movements were awkwardly but correctly executed. In the legs no kind of apractic disturbances were demonstrable. After four weeks of treatment at the clinic there was slight improvement to all appearances, and the patient was discharged. In February, 1920, the patient appeared again in the clinic in substantially worse condition. There was a facial paresis on the left; paralysis of the left arm and leg and excessive spasms in both. The reflexes were greatly increased, the Babinski and Oppenheim signs were positive. The eyegrounds showed beginning papillitis, with swelling on both sides. The phenomenon of the left hand was no more demonstrable. Three weeks later the patent gave birth to a full time child, and five hours afterward died.

The pathologic findings at necropsy were: flattened convolutions and obliterated furrows. On the right half of the brain, in the middle of the gyrus frontalis superior, including the whole of the lobus paracentralis was a grayish-red tumor, arching itself over the median plane and the corresponding part of the left half. This had grown firmly spread out posteriorly, with the falx cerebri. There was found also, on the left side of the falx, a similar tumor the size of a cherry stone. The tumor itself is described as being the size of a child's fist, and of a grayish-red, reddish speckled appearance, owing to the cut surfaces and lumina of numerous blood vessels. Anteriorly the tumor reached to the frontal region and occupied the whole gyrus frontalis superior and the gyrus cinguli, all but a thin strip. The corpus callosum was arched forward to the ventricle so that the anterior horn was severely compressed. The whole anterior central convolution was occupied, except a thin strip on its convexity. The severely flattened corpus callosum lay firmly on the compressed basal ganglions, especially the caudate nucleus, so that only a little space of the ventricle was present at the edge of the fornix; farther posteriorly, also, the gyrus centralis posterior was atrophied on the convexity, all except a thin layer. The thalamus was strongly compressed, the ventricle showing only a slight crescentric lumen. Behind the gyrus centralis posterior the tumor ended, the flattened pole of the egg-shaped structure reaching to the parietal region. Here the lateral ventricles were widened, particularly the posterior horns. The final anatomic diagnosis as given was—large endothelioma of the dura mater.

The author raises the question as to what we are trying to learn from the case described in the foregoing. He classes this as an apractic disturbance of the left hand in the sense of the motor or ideokinetic apraxia of Liepmann. The author cites similar cases from the literature, as, for instance, that of Liepmann, whose patient could not close his hand on command, but in whom the closure succeeded when an object was placed in the hand. The same patient on command could not perform the act of buttoning, but if the hand was placed on the button hole, he smoothly accomplished this act. In all the cases cited voluntary movement of various muscles was not possible, but on being stimulated they could promptly perform their function. Liepmann concluded that the stereotype repeated purposeful movements, such as walking, winking, fist making, to seize or hold firmly an object, as special performances of the sensory-motor mechanism could be satisfactorily performed subcortically, perhaps also spinally, although their peculiar nervous substratum is found cortically in the cortical fields of the various limbs in the form of kinetic memories. This controlling influence of the higher centers as, for instance, the optic and acoustic, is not necessary, as they are performed to some extent through cortical "short circuits" within the sensomotorium dependent on tactile irritation. Granted that the senso-motorium may be completely separated from these higher centers, then the ideatory and the kinetic memories are torn asunder; it therefore follows that the closure into a fist may still result on tactile stimulation as an independent action of the senso-motorium, but not without the proper stimulus or as a repetition (imitation) of an act already performed.

The author states that his case was different from that of Liepmann and others, not only in the fact that a closure of the hand was possible, if an object touched the palm, but that it followed purely reflexly, the patient being unable to inhibit it. This is, according to the definition of Munch-Petersen, a true skin reflex; that is, an involuntary movement which originates through stimulation of the skin and continues; that the skin reflexes are purposeful movements which have as their purpose to remove unpleasant or destructive stimuli from the irritated portion of the body. In this case not only the palm was closed reflexly but also the thumb became active in a very important degree, and produced throughout the impression of grasping or of a claw; therefore the reflex is fitly named the grasp or claw reflex. In the literature the authors found few accounts of similar observations. A palmar reflex, which consists in a palmar flexion of the fingers with irritation of the palm, was mentioned more often than the grasp reflex. Lewandowsky, in mentioning it quite briefly, places the anatomic substratum in the spinal cord—cervical 7 to dorsal 1.

The author seems to be undecided whether the palmar reflex is a preliminary step or an attenuated form of the grasp reflex, or whether it indicates something entirely different. Lewandowsky has observed the grasp reflex in anencephalic patients and Janischewsky has reported two cases almost identical to that of the author. Janischewsky localizes the process in the opposite hemisphere beneath the cortex in the region of the gyrus centralis anterior, and names the process "reflex de préhension."

Strümpell has advanced the following theory concering skin reflexes: These reflexes, even if now appearing to be practically useless, have been in their phylogenetic past most probably attack and defensive movements; they have passed through numerous generations until they have now taken their stereotyped form with a corresponding laying down of anatomic paths. The higher the organization the more they subside and are replaced by voluntary movements. It is therefore no accident that the skin reflexes in the upper extremities are far more unusual to obtain than in the lower. This makes it clear why some reflex phenomena observed in idiots otherwise only appear in infants. According to Doflein, the tendency to grasp spasmodically offered objects in infants is based on atavistic phenomena, and following Strümpell's theory, the loss of the reflex movements is explicable accordingly as useful voluntary movements are acquired.

The author then seeks to localize the site of such reflexes in the brain, since it has been conceded by Munch-Petersen and others, based on recent investigation, that the localization of the reflex center in the cord may have become untenable. Speaking of Oppenheim's feed-reflex, Fürnrohr and others have found a definite area in the cortex whose stimulation produces the characteristic reflex. However, they are led to believe that the real center is in the optic thalamus and that the function of the cortex is simply regulatory and inhibitory. Janischewsky places also the center for the grasp reflex in the subcortical ganglions, probably in the optic thalamus, and he writes that the grasp reflex may be made use of as a diagnostic sign for lesions of the frontal lobe above the subcortical centers (he classes the gyrus centralis anterior with the frontal lobe).

The author concludes that the question of the site of the center for such a reflex is still not definitely decided. This reflex belongs in the series of those which are observed in infancy, but later disappear. Probably the subcortical ganglions would have to come into consideration, and as they are controlled by the cortex, the appearance of this reflex at a later age would, under these hypotheses, speak for a disturbance of the paths from the cortex to the subcortical ganglions.

Wagenhals, Columbus, Ohio

CHANGES IN THE BRAIN IN LETHARGIC ENCEPHALITIS.

Charles B. Dunlap, State Hosp. Quart. **6**:311 (May), 1921.

According to my present experience, lethargic encephalitis is a so-called inflammatory disease with a cellular exudate. This exudate is likely to consist mainly of lymphoid cells with fewer plasma cells or sometimes none, but if any one expects to make his diagnosis on the make-up of the exudate alone he will be disappointed. It is the location of the cellular exudate rather than its character that helps in diagnosis.

In the so-called typical cases of lethargic encephalitis the exudate is seen abundantly in the brain stem, particularly in the midbrain, where the nuclei of the oculomotor nerves are found; it is therefore not strange that paralyses of the eye muscles often occur in lethargic encephalitis. A peculiarity of this exudate is that often it is not confined within the outer sheaths of the blood vessels, as in general paralysis, but, with the vessels as centers of greatest intensity, the exudate is scattered about through the nervous tissues.

The nervous tissues into which the exudate escapes in encephalitis, including the nerve cells, do not seem to be profoundly altered by it, and we have not noticed any marked changes in the neuroglia cells, although occasionally some spider cell reaction and some increase in satellites may be seen.

Another common feature in these cases is great engorgement of the blood vessels, usually with slight local escape of blood into the tissues, so that hemorrhages, of small size, are frequent in lethargic encephalitis, a condition usually not seen in cases of general paralysis or syphilis.

So in the more typical cases of lethargic encephalitis we have, among the cardinal signs of inflammation, redness, swelling and diffuse exudate, especially in the brain stem, with comparatively little of this in the cerebral hemispheres or cerebellar hemispheres.

In the less typical cases, diagnosis with the microscope is much more difficult. In some of these cases we find little exudate in the brain stem, merely a few lymphoid cells around a small number of blood vessels and a few small hemorrhages here and there. The same sort of thing is also found in the gray matter of the cerebral hemispheres, and in the pia mater small bunches or rows of lymphoid cells are seen here and there. The more we looked the more we found in these less typical cases; sometimes, in a case of this kind, a massive lymphoid exudate might suddenly and unexpectedly appear in the microscopic field. For example, in the caudate nucleus of one case a large amount of exudate was found in the walls of only a few blood vessels.

Another rather striking thing throughout the series of cases (both typical and atypical) was a marked swelling of the endothelial cells of the blood vessels and often an apparent increase in number of such cells. These endothelial cells have the property of taking up and disposing of injurious substances and, especially when they get free, they are often actively phagocytic.

In one doubtful case, there were, in all parts of the cortex, small foci of such free endothelial cells around small blood vessels, and the presence of mitotic figures in some of these cells showed that they were actively subdividing. Whether rightly or wrongly, we interpret this as meaning that not only the cells of the exudate, but also the endothelium is active against the effects of the invading foreign material, whatever it may be (globoid organism of Loewe and Strauss?), in lethargic encephalitis.

In trying to differentiate other toxic and infectious processes from what we have called the more diffuse processes as seen in lethargic encephalities, it would seem wise to use both history and microscope, make a reserved differential diagnosis and then leave a loophole for escape.

Except in the most typical cases of lethargic encephalitis, a diagnosis based on histology alone is not safe. There are no absolutely specific pathognomonic features to depend on.

Pollock, Albany.

PERSONAL RESULTS IN THE INTRASPINAL TREATMENT OF NEUROSYPHILIS. B. Rodriguez, Rev. Neurol. **27**:439 (May) 1920.

Rodriguez reports a series of fourteen cases of neurosyphilis in which intraspinal treatment was preceded and combined with vigorous constitutional therapy—intravenous neo-arsphenamin associated with mercurials and potassium iodid. Specifically, in the matter of intraspinal therapy the author utilized both mercurialized serum (in vitro) in dilution of 10 c.c. of serum to 5 mg. of mercuric chlorid and neo-arsphenaminized serum (in vitro) in dilution of 10 c.c. of serum to 5 mg. of neo-arsphenamin (Billon), preferring autoserums where possible, as inducing a minimum constitutional reaction on the part of the patient. It might be mentioned that the paresis cases were noted to be definitely hyporeactional, from this point of view, as compared with the other types of neurosyphilis. The dosage varied, progressively, from 1.5 to 5 mg. in the case of mercuric chlorid and from 2.5 to 5 mg. in the case of neo-arsphenamin, with rest periods of from two to eight weeks.

Comprising the author's series were five cases of general paresis, two of tabes dorsalis, one "cerebral" syphilis, one of cerebrospinal syphilis, four of "spinal" syphilis and one of heredosyphilis with eighth nerve involvement.

Of the first group, in which from two to four subdural injections of mercurialized serum were administered, ranging in dosage from 1.3 to 1.6 mg. at intervals of from two to four weeks, four cases showed transient improvement clinically, with no evidence of amelioration in the fifth, and in all but one, the spinal fluid Wassermann reaction and the colloidal gold curves became somewhat less positive. Of the two tabetics the first, with negative spinal fluid Wassermann and Nonne-Apelt reactions and a colloidal gold curve of 01100000000, showed gradual, but marked improvement after an injection of 1.3 mg. of mercurialized serum, and the second, with feebly positive spinal fluid Wassermann and Nonne-Apelt reaction and with a colloidal gold curve of 11210000000, in which four injections of autoneo-arsphenaminized serum were administered in doses of 2.5, 3.5, 4.5 and 5 mg. at intervals of one and a half months, showed only slight clinical improvement, with some reduction in the colloidal gold curve. The case of "cerebral" syphilis with negative spinal fluid Wassermann and Nonne-Apelt reaction and a colloidal gold curve of 11222300000, showed prompt clinical improvement after one injection of 3.5 mg. of automercurialized serum. In the case of the patient with cerebrospinal syphilis in which the spinal fluid Wassermann reaction was negative, the Nonne-Apelt reaction feebly positive and the colloidal gold curve 11210000000, the administration of three injections of autoneo-arsphenaminized serum in dosages of 2.5, 3.5 and 4 mg. at an interval of one and one-half months, showed slight clinical improvement, negative Nonne-Apelt reaction and slight lowering of the gold curve. In the four "spinal" patients (apparently Erb type) who received two to five injections in doses ranging from 1.3 to 5 mg., of both mercurialized and neo-arsphenaminized serum, there was noted definite clinical improvement and in the heredosyphilitic case, with negative Nonne-Apelt and Wassermann reactions and negative colloidal gold curve, two subdural injections of 1.3 and 2.6 mg. of mercurialized serum were administered at an interval of three weeks with marked clinical improvement. No definite statement is made in these cases as to the permanency of the improvement noted.

In concluding, the author emphasizes the necessity for early, vigorous and persistent, combined treatment in all cases of neurosyphilis, particularly in the paretic and tabetic types, which he found to be, therapeutically, by far the least amenable.

Raphael, Ann Arbor, Mich.

PATHOGENESIS OF CATATONIC STUPOR. H. Berger, München. med. Wchnschr. **68**:448 (April 15), 1921.

Twenty years ago Berger observed that a subcutaneous injection of cocain in a severe case of catatonic stupor produced a transient disappearance of the mental condition. In the present article he presents the results obtained in a study of eleven cases.

In this work the author used from 0.025 to 0.05 gm. of cocain hydrochlorid. Eight of the patients showed decided reactions; three showed no change. The three patients showing no reaction had long standing cases of dementia praecox, while the eight who reacted had rather acute cases of dementia praecox.

In the eight patients the reaction was temporary and frequently the period of mental change lasted only from one to two hours. As a therapeutic measure cocain offers no hope.

Cocain, like other poisons that act on the central nervous system, cannot cause qualitative changes in the central functions, only a heightening or lowering of the normal activity of these centers.

The animal experimentations as carried out by Uyolino, Mosso, and others, have shown that cocain injected in small doses produces stimulation of the cerebral cortex. In man the first noticeable action is a sensation of warmth and a pronounced feeling of well-being. The psychic phenomena are not entirely clear, however. The author used cocain in several cases of manic-depressive insanity of the depressed type and found a heightened psychic activity; thus their time reactions for simple mathematical equations was markedly increased. In one case the average reaction time was 24.9 seconds, while following an injection of cocain the reaction time was increased to 8.8 seconds.

The author does not believe in a psychologic motivating factor in the production of stupor. He believes that catatonic stupor, as a rule, is a primary stupor, so termed by Ziehen, and that it does not disappear by means of psychic influences. Berger does not accept the view of complex formation in stupor as held by Jung.

In the stupor of dementia praecox metabolism is impaired, as are other physiologic functions. The causes for the changes are not easily explained, but, according to Ziehen and others, are due to a marked cortical inhibition. Reichardt believes one must consider a physio-chemical change in the colloidal brain substance, while Bleuler in several cases of stupor has found edema of the membranes with brain swelling.

The author assumes that cocain has a stimulating action on the cerebral cortex. In cases of primary stupor the cortical activity is greatly diminished, and by the injection of cocain there is a stimulation of the "dissimulation processes" with subsequent increase of cerebral function, and as long as the cocain action continues the stupor disappears, to return with the disappearance of the cocain stimulation.

Why the cerebral function becomes impaired is another question. Certainly in the long standing cases organic changes do occur, as is also indicated by their failure to react to cocain stimulation. In agreement with the anatomicopathologic findings, one may assume that changes in the second and third layers of the cortex do not permit the proper function of these cells to take place.

Berger believes that a primary catatonic stupor is an organic syndrome which is due to inhibition or diminished activity of the cerebral cortex. He concludes with the remark that he cannot accept the psychic genesis of catatonic stupor, as a matter of fact of dementia praecox.

Moersch, Rochester, Minn.

SIMULTANEOUS MULTIPLE CLONI IN EPIDEMIC ENCEPHALOMYELITIS. G. Dagnini, Gior. di clin. Med., Pt. 7-8 (July-August), 1920.

By simultaneous multiple cloni the author refers to the manifold manifestations of motor irritation which under the form of involuntary muscular twitching, of different amplitude, localized or disseminated, regular or arythmical, have been observed during the recent epidemic of lethargic encephalitis. He has made a study of these simultaneous clonic contractions in several muscular groups whose action in some cases is asynergic and antagonistic.

In one of the cases tracings of the muscular contractions were taken and compared two by two in the following order: left latissimus dorsi and flexors of the head, right pectoralis major and left flexor carpi radialis, left flexor carpi radialis and flexors of the head, left extensor carpi radialis longus and right pectoralis major, right pectoralis major and elevators of the left shoulder, left scalenus anterior and right rectus abdominis, left flexor carpi radialis and right rectus abdominis. Perfect synchronism of the muscular contractions was demonstrated in every one of the muscles examined, including the respiratory muscles. The contractions ceased during sleep, and could not be influenced by the patient's will. Emotions, suggestion, peripheral stimulation or bromids did not modify the intensity, the frequency, the rhythm and the localization of the cloni. Synchronism was not accidental.

The author points out that in these cases, unlike those of epileptic seizures, of tic convulsif and also of chorea, in which the synergic movements originate from a contemporaneous stimulus acting on centers having reciprocal anatomic and functional connections, it is difficult to interpret the appearance of simultaneous clonic contractions in muscular groups whose centers are not in direct relationship anatomically or functionally.

What cortical, subcortical, spinal, radicular and peripheral relationship exists —the author asks—among so distant and functionally different muscles, whose centers react contemporaneously to the same stimulus? Supposing that an abnormal stimulus, starting from a slight lesion in the rolandic zone, caused contractions in two muscles of the forearm, why were not the other nearby centers for the upper left extremity involved, whereas more distant centers (for the elevators of the shoulder, latissimus dorsi, scaleni, pectorales, diaphragma) and even contralateral centers were irritated? If the phenomenon were of spinal origin, why did cellular groups belonging to various segments from the cervical cord (flexors of the head) to the dorsal and lumbar sections (rectus abdominis) react simultaneously to a stimulus without the near and the intermediate zones being involved?

The author propends in favor of a spinal origin. Small symmetrical lesions of the anterior horn cells may explain better the phenomenon, not that symmetrical movements cannot originate from unilateral lesions of the brain, but because the succession of the contractions suggests the existence of two distinct lesions localized in homologous seats. The stimulus must have different effects according to the degree of excitability of the cellular elements on which the identical stimulation is successively exerted. Probably a condition of increased excitability of each clonogenous cell plays an important rôle in circumscribing within given zones the effects of a stimulation which may also irradiate elsewhere, if the recepting cellular elements have not reached a sufficiently high reacting power.

There may be an analogy with the systemic lesions of the spinal cord, in which probably the bilateral and uniform morbid localization is dependent on chemical affinity of given poisons for groups of cells or fibers symmetrically disposed and equally subject to the same pathogenetic cause.

Naccarati, New York

A STUDY OF THE SPINAL FLUID IN ONE THOUSAND EIGHT HUNDRED AND SIXTY-NINE CASES OF SYPHILIS IN ALL STAGES. U. J. Wile and C. H. Marshall. Arch. Derm. & Syph. **3**:272 (March), 1921.

The authors quote a previous communication of their own by restating that the invasion of the nervous system in syphilis occurs extremely early—even seriously—without symptoms, and undetected except by means of spinal fluid examinations. They believe that cerebrospinal involvement, when occurring, does so within the first few months of the infection. The conclusions based on a study of the 1,869 positive cases herein reported are that if the nervous system is not involved early in the disease it is not often involved later. Furthermore, they view the high percentage of positive findings in primary and secondary syphilis as in the nature of a meningeal roseola and transitory in character. On the other hand, a certain percentage of cases is found in which the cerebrospinal involvement is considerable but shows few signs or symptoms; these cases are in the latent period and may become symptomatic later. In the tables accompanying the text it is shown that there is considerable parallelism in the percentage of positive findings.

Percentage of Positive Findings

Type of Syphilitic Involvement	Number of Cases	Percentage Increased Albumin and Globulin	Percentage Increased Cells	Percentage Positive Wassermann Reaction
Congenital	53	13.2	26.4	16.9
Primary	236	19.0	16.1	5.9
Secondary	508	35.1	34.0	26.5
Tertiary	155	26.4	25.1	30.9
Latent	568	27.1	28.6	27.9
Diffuse cerebrospinal	178	97.2	88.2	87.6
Tabes	109	98.1	93.5	93.5
Paresis	49	95.9	95.9	98.9
Taboparesis	13	100.0	100.0	100.0

By reference to the consolidated table, it is noticed that of nervous system involvement, cerebrospinal syphilis is lowest in uniformity of positive findings. The authors deal briefly with the frequency of association of neurosyphilis with syphilitic alopecia, iritis, leukoderma colli and late syphilids of the skin. There is a high percentage of asymptomatic neurosyphilis cases during the period of latency.

The paper is very interesting from the statistical point of view, and it is regretted that the colloidal gold curves could not have been made in each case to complete the data.

Patten, Philadelphia

SOME PRACTICAL POINTS IN THE ORGANIZATION OF TREATMENT OF SYPHILIS IN A STATE HOSPITAL. AARON J. ROSANOFF, State Hosp. Quart. **6**:319 (May), 1921.

The cases of syphilis which are encountered among state hospital admissions may be classified as follows:

Systemic syphilis—latent; active. Neurosyphilis—mesoblastic (cerebral) syphilis); parenchymatous (general paralysis).

As regards latent systemic syphilis, the main point is that it can be brought to light only by means of routine Wassermann tests made on all patients admitted to the hospital. There is, as a rule, no etiologic connection between the syphilis and the mental disorder in such cases. The patients should, however, receive antisyphilitic treatment on general principles, and, in event of parole, provision should be made for continuance of such treatment extramurally.

As regards cases of active systemic syphilis, it need hardly be said that these patients are in even more urgent need of prompt, vigorous and persevering treatment, although in such cases, too, there is no etiologic connection between the syphilis and the mental disorder.

In cases of mesoblastic invasion, we find, as the most significant of the recently discovered facts (1) that such invasion occurs in a high percentage of cases early in the course of the infection, namely, in the primary and secondary stages, and (2) that it may exist without giving rise to nervous or mental symptoms, or may manifest itself by slight, vague, or transient symptoms, so that there is danger ot its being overlooked.

The practical bearing of these facts is that all patients with systemic syphilis, latent or active, should be, at some time in the course of their treatment, investigated by lumbar puncture, in order to dispose of the question of possible invasion of the central nervous system; and that no patient with syphilis can be discharged as cured until not only the clinical and blood serum findings have been rendered persistently negative but also the spinal fluid findings.

We now come to the consideration of neurosyphilis caused by parenchymatous invasion, i. e., general paralysis. In this connection the question is perhaps to be formulated as follows: Considering all past experience, which is discouraging, should such patients receive antisyphilitic treatment? In my opinion, the answer must be in the affirmative.

There has been much discussion as to the choice between arsphenamin and neo-arsphenamin. The question presents for consideration three distinct elements: (1) trypanocidal power, (2) toxicity, and (3) convenience in administration.

Experimental investigations recently made by Schamberg and his associates in the Dermatological Research Laboratories of Philadelphia have shown that arsphenamin is one and three-fourths times more curative than neo-arsphenamin, but is at the same time two and one-half times more toxic. Accordingly, it is possible to choose for neo-arsphenamin such dosage as to secure greater curative and less toxic effect than by means of arsphenamin.

The technic of administering neo-arsphenamin is by far the simpler for the physician and the less uncomfortable for the patient. The ampule is opened by filing and breaking off the pointed end; the neo-arsphenamin is dissolved in the ampule with about 5 c.c. of freshly distilled water at room temperature; and the solution is slowly injected intravenously with a 10 c.c. glass syringe. A dose of 0.9 gm. should be given in not less than 10 c.c. of water. The ampules in which the neo-arsphenamin is furnished generally have a capacity of only a little over 5 c.c., but more water can be drawn into the syringe to dilute the solution before it is injected. Higher degrees of concentration, especially if injected too rapidly, cause hemolysis, which gives rise, within twenty-four hours, to a reaction characterized by chills, nausea, vomiting, headache, pains in the arms and legs, some general prostration and occasionally slight elevation of temperature.

Pollock, Albany

THE DIFFERENTIAL DIAGNOSIS BETWEEN MYOCLONIC UREMIA AND EPIDEMIC ENCEPHALITIS. Henri Roger and André Chaix, Presse méd. **29**:461 (June 11), 1921.

Two cases of uremia with myoclonic symptoms are reported in detail and compared with others in the literature. Some fairly definite cases of myoclonic epidemic encephalitis have shown pathologic kidneys at necropsy, so that the symptom in question could not be ascribed to either cause exclusively. Clinical differentiation may also be difficult. Epidemic encephalitis is responsible for the majority of such cases, while toxic conditions other than uremia can also give rise to a similar syndrome.

Uremia should show characteristic clinical signs, such as vomiting and epistaxis, and give a history suggestive of renal insufficiency; but without these the absence of fever and pains when uremia or encephalitis is suspected is in favor of the former. Epidemic encephalitis of the myoclonic type nearly always causes a febrile reaction, at least at the beginning, while pains are usually noticeable and often severe. One must remember that hiccups and lethargy are uremic as well as encephalitic signs.

The cases of myoclonic uremia were characterized by sharp contractions and twitches with little movement at the joints, attacking limbs, face and abdomen indiscriminately. These contractions were sometimes choreiform. Reflexes were usually exaggerated, with clonus.

A lumbar puncture yielding evidence of meningeal irritation does not necessarily exclude uremia. Neither does a slightly increased urea content of cerebrospinal fluid and of blood exclude encephalitis. It has been claimed that histopathology could furnish positive differentiating evidence: that degenerative or inflammatory changes found in encephalitis would not be found in uremia. These writers, on the contrary, expect that central nervous lesions will be demonstrated.

Uremia with generalized myoclonic contractions offers a bad prognosis; if the symptoms are localized the prognosis will be better. Treatment is the same as for any other form of uremia.

Besides myoclonic uremia, four other nervous types are described: eclamptic, jacksonian, tetanic (disputed by some authors) and ataxic.

Huddleson, New York

ABNORMAL LOCALIZATION OF TETANY IN THE ADULT. Victor Cordier and Auguste Gonnet, Rev. Neurol. **27**:244 (March) 1920.

Cordier and Gonnet report a case peculiar, or of the fruste type, in that only one leg was involved, but that typically, directing attention, at the same time, to the scant recognition accorded such apparently aberrant forms in the literature, in spite of their comparative frequency clinically.

The case was that of a man, aged 67, a cabinet maker, who complained of shortness of breath, noted for five years past, and painful cramp-like attacks affecting the left leg, first noted about two years previously. In regard to the latter it was reported that the attacks occurred at intervals of from about eight to fifteen days and could be controlled only by continued rest in bed in the extension position. There was a history also of similar attacks affecting the fingers six years previously as the result of which the patient was no longer able to use his joiner tools. There was no history of alcoholism and no clinical evidence of syphilis.

Physical examination showed well marked emphysema, chronic bronchitis and a blood pressure of (systolic) 160, (diastolic) 80, but was otherwise essentially negative.

Neurologic examination, during one of the "leg attacks" showed the anterior crural and quadriceps femoris muscle groups, of the left leg, to be in tonic contracture with the toes, particularly the hallux, in hyperextension and the knee somewhat elevated. The other muscle units were not involved. This contraction phase was associated with excruciating pain of a local and cramplike nature. Relaxation occurred in approximately ten seconds and was succeeded by another phase of contraction which lasted about four or five seconds and so on for two minutes. Stimulation of the muscle elements involved was found to precipitate a typical contracture phase, but no change was noted on stimulation of the other muscle groups. Electrical examination showed hyperirritability of the tibial and common peroneal trunks. Neurologic examination was otherwise completely negative.

Etiologically the authors seem inclined to discount the importance of paratyphoid disturbance in many cases of tetany, so-called, and on the basis of the case presented suggest the possible significance—direct or indirect—of occupational posture-tensions.

RAPHAEL, Ann Arbor, Mich.

BRAIN AND THYROID FUNCTION. C. CENI. Riv. sper. di reniat. **44**: 243-286 (July), 1920.

Ceni has studied the effect of partial and total removal of the brain on the function of the thyroid gland.

In lower vertebrates (*Chelonia*) no relation was found to exist between the forebrain and the thyroid function. The total extirpation of the brain produced neither immediate nor later changes in the thyroid.

In birds (fowl and pigeon) decerebration and hemidecerebration was followed by diffuse colloid degenerative atrophy within twenty days from the operation.

In the same animals, dead or killed from thirty-five days to three years after the operation, the thyroid alterations were more pronounced; they involved all the thyroid structures (colloid, alveoli, septums, epithelium, vessels), assuming the character of diffuse parenchymatous and interstitial hypertrophy.

In fowls no definite relation was found to exist between the states of atrophy and of hypertrophy of the thyroid gland and the general development of the body and of the external sexual characters. No constant relation was found between the hypofunction and the hyperfunction of the thyroid gland and the hypofunction and hyperfunction of the sex glands.

In dogs the removal of the cortex of one cerebral lobe or of the surface of one cerebral hemisphere generates thyroid alterations similar to those found in birds, namely, diffuse colloidal hypertrophy, which may also reach the degree of parenchymatous hypertrophy. These phenomena, likewise in birds, are not constant, but are frequent and vary in degrees in divers subjects.

The general conclusions which the author draws from his experiments are:

1. In vertebrates exist superior thyroid centers, which in birds are disseminated in the mass of the whole forebrain, especially in the striate body, and in mammals are distributed in the entire cerebral cortex.

2. The superior thyroid centers have a trophodynamic inhibitory function, which, in opposition to the accelerator function of the genetic centers, act by preventing an excessive function of the peripheral organ, by regulating the circulation and by maintaining in normal proportions the trophic processes of the different elements, especially of the parenchymatous ones.

The author points out that the results of his experiments call for a revision of many clinical problems, especially in neuropsychiatry, in view of the present tendency on the part of clinicians to interpret any alteration of the thyroid gland as a cause rather than as an effect of the clinical picture.

Naccarati, New York

REGARDING A NEW SUPERFICIAL REFLEX—THE PALMO-MENTAL REFLEX. G. Marinesco and A. Radovici, Rev. Neurol. **27**:237 (March) 1920.

The authors describe an additional reflex of the cutaneous series for which they propose the designation palmo-mental reflex.

This reflex, which the authors have found to be particularly characteristic in pyramidal tract lesions, especially when bilateral, consists in a slow and enduring contraction of the superficial chin muscles (triangularis, quadratus, labii inferioris and transversus menti) ipsolaterally, following stimulation of the palmothenar area. Care must be taken in eliciting this response that the chin muscles be relaxed and the mouth held slightly opened; gross posture is of no primary importance as the reflex may be elicited equally well with the patient sitting or in lateral decubitus. It may be remarked that in pathologic cases this mental response may be secured from the proper stimulation of practically any sensory area, but seems most delicate, in the authors' experience, on stimulation of the thenar surfaces as described.

This response was also noted in 50 per cent. of a series of 300 normal subjects (particularly in infants), but differed from the reaction determined in pathologic cases in that the contraction was definitely quicker, of shorter duration and that the response was noted only after stimulation of the palmothenar surfaces and did not follow excitation of other sensory areas.

This reflex is found to be abolished in the presence of first motor or sensory neuron lesions, thus being of interest in the differentiation of functional and organic disorders. The mechanism of response is not wholly clear although the authors feel it to be mediated, indirectly, by intercalated neurons through the lateral columns and thence, by collaterals, to the facial nuclei, ipsolaterally.

Raphael, Ann Arbor, Mich.

THE SUBSTANTIA NIGRA: A BRIEF COMPARATIVE ANATOMICAL STUDY. I. S. Wechsler, Neurol. Bull. **3**:130-134, 1921.

The author reviews the literature on the substantia nigra and gives the results of his own study of serial sections of seven simians, two other animals and man. He states that the structure is about the same in all mammals, though it is best developed in man. It is not well defined and cannot be sharply delimited from the tegmentum, internal geniculate body, pontile nuclei or the hypothalamic region. It contains four groups of cells. The fiber connections are not thoroughly worked out, but as nearly as can be determined, it is connected with the frontal, temporal and central cortex, with the caudate and lenticular nuclei, the internal geniculate body and the spinal cord. Whether these fibers are centripetal, centrifugal or both, has not been definitely decided. The substantia has not been studied in birds, and is said not to exist in the lower vertebrates. Bauer believes that it was originally an undifferentiated structure and that its cells have developed from the central gray and moved ventrally, which explains the reason why some of the cells are found in the lemniscus and reticular substance. Its function is summarized in a paragraph from Bauer, as follows: "Just as the numerous cells grouped throughout the brain-stem and conceived by Edinger as the nucleus motorius tegmenti are under the influence of the cerebellum, so the cells of the substantia nigra are for the purpose of coordination under the influence of the cortex. The cells carry impulses from cerebral centers, from the cortex or corpus striatum, directly or through association cells of the tegmentum, to various efferent cell complexes. The coordination of chewing and swallowing is only one of the numerous associational and coordinative functions of the substantia nigra."

Howe, New York

PLACENTAL TRANSMISSION OF EPIDEMIC ENCEPHALITIS.

R. Mercier, in collaboration with Andrieux and Bonnaud, Bull. de l'Acad. de med. **85**:625 (May 31), 1921.

A young woman, aged 28, twenty-two days before delivery of a child, developed epidemic encephalitis, with pains and myoclonia at the onset and subsequently diplopia, abolition of reflexes and delirium.

The spinal fluid was clear, without bacteria or lymphocytosis. There were appreciable traces of albumin and of sugar. The Wassermann test was negative.

At labor, though dilatation occurred, uterine contractions were deficient and delivery by forceps was necessarily resorted to.

There was no subsequent uterine infection. However, the labor appeared to act as a traumatic shock and greatly intensified the encephalitic symptoms, and the mother died in coma on the fortieth day.

The infant, who was from the beginning separated from the mother, showed myoclonic movements of the head and upper extremities, attributed to an encephalitis. These began in the second week of life and gradually disappeared over a period of three weeks. This condition was not accompanied by fever. Residuals in the child have been a certain nervous excitability and an occasional convergent squint. Otherwise, the child thrives.

These clinical facts are reported as well proving the transmission of epidemic encephalitis by the placenta.

Davis, New York

ISOLATED NEURITIS OF THE MOTOR BRANCH OF THE TRIGEMINAL NERVE.

K. H. Krabbe, Rev. Neurol. **27**:241 (March) 1920.

Krabbe, directing attention to the rarity of isolated neuritis of the motor component of the trigeminal nerve, makes report of a case in which difficulty in opening the mouth and in mastication was noted several days after the onset of what appeared clinically as a typical attack of influenza with bronchitic features.

The systemic condition apparently subsided speedily and uneventfully, but the neurologic disturbance was noted to persist in diminishing progression for some months, localizing principally to the right with some flattening of the face on this side.

On examination by the author, ten months after the disturbance had first been noted, there was remarked pronounced atrophy of the right temporal and masseter muscles with marked weakness in the right bite. There was also indication of paralysis of the right internal pterygoid as it was impossible for the patient to draw the mouth to that side. The right corneal and masseter reflexes were found to be diminished, but no frank sensory deviation could be determined and no disturbance could be discovered in the fields of the other cranial nerves.

The author makes no mention as to the ultimate outcome in this case.

Raphael, Ann Arbor, Mich.

THE MENTAL CLINIC AND THE COMMUNITY. Everett S. Elwood, State Hosp. Quart. 6:381 (May), 1921.

The establishment of after-care work at the Manhattan State Hospital was the first definite move in this direction in the state of New York. This work has grown so that the state hospitals now have a total of twenty-three social workers and forty-one clinics. As the people become more and more acquainted with it their prejudice and superstition against all forms of insanity gradually disappear.

The work of the mental clinics is greatly appreciated by most of the communities in which they function. During the last year we have received requests from several cities for the establishment of clinics under the guidance of state hospital physicians. The dean of a prominent medical college recently asked what he could do to facilitate the organization of this good work in his city. Public school departments are particularly anxious to receive the help that clinics afford in the diagnosis and treatment of borderline cases developing among schoolchildren.

The time is probably not far distant when each state hospital will have to have an extra physician on its staff to attend to this field work, for the assignments of duties within the institutions are in most instances sufficient to demand the whole time and energy of the medical staff.

Such a service to the community should have the adequate support not only of the state hospitals themselves and the various social forces in the different communities but also from the appropriating authorities of the state. It is not only a saving in dollars and cents to keep a patient sufficiently balanced to forego the necessity of hospital commitment, but it is a great saving in the welfare and happiness of the individual patient and his family.

Pollock, Albany.

CLINICAL AND ANATOMOPATHOLOGIC CONTRIBUTION TO THE STUDY OF HEREDITARY ATAXIA. G. Mingazzini and F. Giannuli, Gior. d. r. Accad. di med. di Torino, 1919.

The patient who forms the basis for this anatomopathologic study was suffering from spinocerebellar ataxia. She had been for many years under the author's observation. The interest in this case is increased by the fact that

Mingazzini had studied in the same manner a brother of the patient several years ago. The conclusions reached by the authors in this research confirm the previous work of Mingazzini.

The present investigation has been extensively and accurately carried out with serial cuts of the brain and of the spinal cord; the latter has been studied with greater riches of particulars of the degenerated areas. There are thirty-four figures in the text.

One of the conclusions confirms the pseudosystemic type of the degenerative processes in this disease.

Another important conclusion regards the behavior of the glia. The authors demonstrate that glial alterations are found, not only in the spinal cord, but also in the cerebrum and in the cortex; such alterations follow the same phases and show the same characteristics observed in cases of disseminated sclerosis of the brain.

Finally, the authors, basing their assumptions on their histologic findings and on those of previous investigators, maintain that cerebellar ataxia is not a disease limited only to the cord and cerebellum, but that it represents a morbid condition affecting the whole spino-cerebro-cerebellum system.

The thesis advanced by the Roman school has been emphasized in one of Mingazzini's recent papers, in which he reiterates the fact that spinocerebellar aplasias, as well as some other diseases hitherto classified as purely spinal, are to be considered as cerebrospinal diseases.

Naccarati, New York

NEW RESEARCHES ON THE MORPHOLOGY AND BEHAVIOR OF THE CONSTITUENTS OF THE MYELIN SHEATH IN SECONDARY DEGENERATIONS. F. Guidi, Riv. di patol. nerv. **26**: Pts. 1-2, 1921.

The author refers to his previous works on secondary degeneration of the fibers of the sciatic nerve, published in the same journal, in which he demonstrated that the two elements of the sheath (granules and alveoloreticular stroma) are a biologic reality, not artificial staining products, as they do not appear at the same embryogenetic date, but the granules can be demonstrated only many days after the stroma.

In this new series of researches carried out with the same method (author's modification of Besta's method), which he describes in full, the author endeavors to integrate his previous study in order to answer some important questions left unsolved, namely: What are the reciprocal relations governing the granules and the stroma in secondary degeneration? What are the morphologic characters of the degeneration and of the disintegration of the granules? Since the two constituents of the sheath appear at different embryologic dates, will they disappear also at different dates? How is the degenerated material absorbed?

Here are the conclusions at which the author has arrived in this new experimental study which was carried out on the sciatic nerve of rabbits at different dates.

The granular portion of the myelin disappears earlier than the alveoloreticular stroma; it assumes special modifications (metachromatic staining) in the affinity for the staining substances used.

During the degenerative process, the granules show phases which morphologically remind one of the behavior of the stroma; that is the granules become grouped and fused together, the fibers become irregular in their course, are broken up and gradually disappear. The granules disappear more rapidly than the fragments of the alveoloreticular stroma. The metachromatic staining takes place chiefly in the first phase. The elimination of degenerated granules seems to take place by means of nucleated elements possessing phagocytic powers in which granules are included, probably in order to be directed toward capillaries.

The author is unable to speak definitely concerning the origin of these nucleated elements. He has undertaken a special research on this subject.

NACCARATI, New York

Society Transactions

CHICAGO NEUROLOGICAL SOCIETY

Annual Meeting, May 27, 1921

Dr. G. B. Hassin, M.D., *President, Presiding*

A PSYCHOSIS ASSOCIATED WITH EPIDEMIC ENCEPHALITIS.

Dr. D. Rotman.

A negress, 28 years old, was admitted to the Chicago State Hospital from the Psychopathic Hospital, April 2, 1921. She had marked tachycardia, was extremely restless and profoundly exhausted. Her temperature was 101 F. on admission and had been about 100 F. ever since. The pupils were fixed and angular. There was some misalinement of the eyeballs, hardly enough to be called strabismus. The knee jerks were overactive; there was no Babinski sign. These findings were still present. During an infection of one arm, with a temperature of 104 F., a marked and regular twitch of the left side of the face and of the arm was noted.

The patient had been admitted to the Cook County Hospital, January 1, with marked pain in the shoulder and neck and tremor of the left arm, the same arm that later showed the motor irritation during the arm infection. A tentative diagnosis of neuritis was first made but the appearance in rapid succession of double ptosis, diplopia, lethargy and other symptoms, led to an early diagnosis of epidemic encephalitis. The temperature ranged from 99 to 102 F., the fluid showed 20 cells, the Wassermann reaction was negative. No colloidal gold test was made. Recently both blood and spinal fluid Wassermann reactions had been negative on two occasions; the colloidal gold curve was 1244321000; there were no cells.

Apparently visual hallucinosis and delusions were present from the beginning. She exhibited great fear, thought she was dying and about to pass into hell where she was to be chased by snakes and various animals. On several occasions she failed to recognize her husband.

After nine weeks in the Cook County Hospital, the patient was discharged as convalescent on March 8, but almost immediately she developed marked mental symptoms resembling the preliminary expansive state of a paretic. She believed that she and her family were to visit the folks in the South. They were to do things in grand style. Everybody was to have a new outfit. Asked where the money was to be obtained for all of this, she stated that God had placed the necessary funds in the bureau drawer. She further stated that she was in direct communication with God, and He had told her that if her orders were not carried out the world would be turned topsy-turvy. Taken to the Psychopathic Hospital, she was very much excited, uncooperative and resistive. Since that time the mental symptoms had been ever-changing.

Since admitted to the state hospital she had been disoriented. Unclearness and clouding had been present at all times but had been so fluctuating in degree as to suggest delirium. For short periods she had known the place to be a hospital. At times she had recognized her physician as such, but most of the time she felt herself to be in her home environment. She called the attending physician Plummer, had never called any one else by this name and had never used any other name for him. One of the nurses who had been with her she called Alice and by no other name, nor did she call any other nurse by this name. This was done by a patient supposedly in delirium.

When first admitted she spit on every one who came near her. This spitting was forceful and seemed purposeful and she would contrive to make it effective. Her explanation was always the same: "I spit on you to save your life." She attacked those about her very much after the fashion of an epileptic in a furor. Unlike an epileptic, however, she was not amnesic for these outbursts. Recently a carpenter had been on the ward making repairs. She forced herself into the room where he was working and was about to snatch the hammer from his hand to strike him. She had remembered this and had mentioned it from time to time with regret, saying "Alice, why did you not let me kill that carpenter?" Some time ago she had broken away from the nurse, had gone to a window, had broken a pane and picked up a piece of glass ready to strike any one who came near, and it was necessary to rush her with a mattress before the glass could be taken away from her. As a matter of fact, this patient had never been physically subdued. Physical restraint had not been successful. She had chewed away the sheets that tied her to the bed and got loose. She had no explanation to offer for these furious attacks.

At times she had remained quiet and seemed to be in a stupor. While in one of these quieter moments a violinist played the chords of "Old Black Joe" for her. She cried immediately and said it reminded her of her father. She then insisted that the musician was her father and called him by name. She then changed from father to son and insisted that the musician was her son. She called to him during the ten minutes he was in the room and begged him to untie his poor mother. This showed how the patient in her delirium was able to weave in some of the things in her surroundings. It also illustrated the peculiar mechanism whereby in her weaving she stopped suddenly and clung to a certain false idea.

A CASE OF ANXIETY NEUROSIS WITH OBSESSION. Dr. I. B. Diamond.

A young woman, aged 22, an intelligent stenographer, was in bed when first seen because of attacks of palpitation, dizziness and a "sinking feeling as if about to die." The attacks greatly frightened her. She had been nervous for three years, beginning when her mother was very ill. Following this was great disharmony with her office associates and dissatisfaction with her job, which, however, she would not give up because of the liberal pay and easy hours. Then came discontent because she was not married (as were many of her friends) and hopelessness of a good match because so many of the eligibles were in the army. It was easily ascertained that she was sensitive, romantic and a day dreamer. Later and with greater difficulty it was learned that a popular movie actor had been her beau ideal. His physical traits and love making had touched her deeply. She never missed one of his films and identified herself with the heroine with much erotic pleasure. But when she learned that he was much married and the father of numerous children she was disenchanted and gave up his pictures.

Recently she had become engaged and avowed supreme satisfaction with her fiancé. Especially she liked his steady and sedate ways and lack of familiarity. Consequently she was much disturbed by a dream of him; the first dream related to Dr. Diamond. He entered her room disguised as a villain or thief and his sinister expression so frightened her that she awoke. Then it was learned that a previous (rejected) admirer as a parting shot had said "beware of a Spaniard" and that her fiancé had spent a large part of his life in Spanish countries. Ideas connected with the dream were indicated by the words villain, treachery, Spaniard, seduction, sexual assault. By degrees she admitted that her lover was too close with his money, and that his personal appearance did not please her.

Then she related the following dream: She was out walking with a girl who reminded her of a girl of the neighborhood who had a bad reputation and with whom she had never associated. They bought apples and ate them. Then she went home, was to be dressed in white and was about to die. "Whatever I was going into was regular death to me, against my will." Apparently she was much agitated by this dream and Dr. Diamond's interpretation was that "she is going into or going to do something bad which she fears; namely, marriage, which will lead to her death." Consequently the dream increased her nervousness. After reassurance and explanation that the dream was only symbolic she was much better for a short time. Soon she was in bed again, afraid of consumption and insanity. Now with reluctance she confessed to the obsession that her fiancé had negro blood. When with him she was comfortable, when absent the imperative idea acutely distressed her. Frink (Morbid Fears and Compulsions) says that for American girls the negro is a symbol of sexuality.

Soon after this the patient related another dream. A Chinese woman with a little boy came to the house and wanted a drink of water. Her mother would not let them in, but the patient said "let me get it for her." Then the woman's husband, a big, fat Chinaman, came in, took the patient in his arms and squeezed her. She threw him to the floor and asked her father to put his foot on his neck, but her father (of whom she was exceedingly fond) was indifferent.

Two further episodes in the history were now brought to light, the first apparently forgotten, the second dissembled. Some years before she and another girl in the office, on leaving the ladies' toilet had been confronted by an exhibitionist entirely exposed. They ran away in terror. She recalled that the expression of her lover in the first dream reminded her of this man's expression. It was not true that her fiancé had been uniformly staid and correct. Soon after their engagement she was greatly shocked suddenly to find his hand in her bosom. She was indignant; on his knees he begged her pardon and never again transgressed. Dr. Diamond comments, "it was evident that . . . she had a great fear of the gross sexual (repression) and probably this was the cause of her neurosis." His summary follows.

"We have here a very sensitive and imaginative girl indulging a great deal in romantic day dreaming who received a shock by an exhibitionist. This brings her in conflict with her dream life which she represses with the resulting neurotic symptoms. Her symptoms disappear as she reverts to day dreaming, this time with the help of a motion picture actor. She becomes shocked a second time to find her hero but an ordinary mortal. Her psychic love life is again repressed followed by the return of her symptoms. Unable to make adjustment, she therefore gets into various difficulties in real life. Although 'sick of heroing' as she expressed it, her interest has not been entirely eradicated. The real heroes being sent to war, she therefore regresses to a childish level by her attachment to her father (substitution) which later influenced her love choice. She now receives a third shock by the too ardent wooing of her lover and develops doubts and fears and later obsessions as a result of her conflicts. Through analysis she obtains a clear insight into her trouble and then makes adjustment to normal which fact she well appreciates. Her comment is worth repeating, 'Doctor, wasn't I the big, silly goose?' It is now almost two years since her illness with no return of her symptoms. She is married and a happy mother."

DISCUSSION

Dr. Clarence A. Neymann looked on the dream essentially as a wish fulfilment. He saw three parts: first, the man who came in with the idea of inflicting some injury; second, the dream of everybody to be feared; third, the dream of the Chinaman, in which the father did not come to her rescue. It seemed to him that the wish fulfilment was not because her lover went too far, but not far enough. He thought Dr. Diamond was dealing with a libido which had not been observed and to which she had not given expression.

Dr. Ralph C. Hamill thought the first dream was of much interest. One frequently meets, in a young girl, the wish for some sexual attention, but she does not want to be responsible for it. In the first dream the man came in as a thief, a housebreaker who was going to do something to her. Dr. Hamill's criticism fell in the same line as Dr. Neymann's; namely, that such a dream indicates a wish for further procedure and yet, perhaps, the more important side was that the patient was not willing to accept responsibility for the wish.

Dr. Meyer Solomon thought that fundamentally the patient had a neuropathic constitution, and that if one went over her life history carefully it would be found that some of the characteristics were instability, a tendency to undue suggestibility, a constant struggle to come to conclusions about life, and, finally, with her meager knowledge to accept these conclusions as facts. She was at the age where sex problems were not the big thing, and she looked at them as at other things. She had a tendency to flee from life in general, not only from sex problems. The analysis aided her to handle her sex problems directly and to get hold of life more directly. Dr. Solomon wondered whether her recovery was due to the technic that was followed, or whether the analysis did not open up for her a realization of the few defects she had.

Dr. Hugh T. Patrick believed that in this case, as in others of successful psychanalysis, it was not necessarily because the psychanalytic conclusion was the true answer that the girl recovered, but because something was done which satisfied the patient, gave her a new start and helped her to adjust herself. He had no doubt that Dr. Neymann with his interpretation and Dr. Hamill with his conception of the psychology and Dr. Solomon working on the neuropathic constitution, might have been just as successful with the patient as was Dr. Diamond, and it might have been that some old-fashioned doctor, without asking her anything about her dreams, might have gotten along just as well as they with their psychanalysis. He thought the most pertinent thing in the history of the case was the sentence at the end of the paper. "She is married and a happy mother."

Dr. Sydney Kuh said that there had not been more different opinions than speakers, but that he wished to give his interpretation of the case. He felt there was one element back of the case which Dr. Patrick had not mentioned. Dr. Diamond analyzed the girl and explained that all her troubles were due to the nervous instability, but Dr. Kuh thought probably there was a certain hysterical element. She got the impression that this process would relieve her of all her troubles and therefore responded to the treatment.

Dr. William G. Stearns suggested that had the young woman fallen into the hands of an expert osteopath she might have obtained the same results. He believed that all the patient needed was an explanation and assurance that she would get well; that any explanation that would appeal to the patient's sense of logic would cause the same result.

Dr. Julius Grinker said that in a case similar to Dr. Diamond's the patient told him that she was sure that if she had a child she would die, and therefore she had prevented pregnancy. A blind physician had listened to her heart and told her she should not have more children. Dr. Grinker examined her heart and told her that as he could not only hear but also see, he was in a favorable position to assure her definitely that she had no heart lesion; this was sufficient to cure the woman. The patient was not obliged to visit him for weeks and months. Dr. Grinker believed that whatever results Dr. Diamond obtained in his case were due entirely to suggestion. He believed that one must use psychanalysis when examining these patients, examine the workings of their minds and urge them to tell everything that troubles them, and when the physician can throw light on the obscure corners of their psyche and suggest rational modes of viewing a situation—his patient will be cured.

Dr. I. B. Diamond (closing), in reply to Dr. Hamill said that the patient really had a wish for sex attention but was absorbed with the psychic love-life rather than with the physical. The exhibitionist had disgusted her along the physical side, and she was afraid of the whole situation. In the first dream she could not understand why she was so much afraid. He was not thinking of the sex question particularly; the girl was romantic and he had to remove the fears before he could get down to the main complex.

In reply to Dr. Stearns, he would state that the girl had been treated by a gynecologist with tampons for some time and had been treated by a neurologist who tried suggestion, but the patient did not respond to either method. She was cured by obtaining an insight into the cause of her trouble, enabling her to make the adjustment.

OCCUPATIONAL THERAPY IN THE STATE HOSPITAL. Dr. E. A. Foley.

Dr. Foley showed numerous pictures illustrating the work of the occupational department of the hospital.

DISCUSSION

Dr. H. Douglas Singer, state psychopathologist, said that his experience with occupational therapy went back to 1902, but he thought something new had been developed with the work in Illinois. That concerned a realization of what one was trying to do with a better organization. The majority of physicians in thinking about occupational therapy considered it only as something to divert the patient's mind. That was really the smallest part of the present work. He had been in the habit of subdividing the measures used in occupational therapy into three classes:

First, "diversional," in which the effort was merely to direct the mind and keep the patient occupied. It is in this form that it enters largely into the work of a general hospital.

Second, not now used to the extent that it might be, was the form of occupational therapy which he called "symptomatic." As an illustration, there was the man who smokes. Men use smoking to rest themselves. When it becomes a habit it does not have much toxic effect, but is a form of monotonous activity. Ladies usually take up some other monotonous activity such as knitting, a form of work that requires little attention. There was no question in his mind that the same effect can be obtained by similar means to produce quiet and rest among excited patients. He had known many patients who had discovered this for themselves. Many excited, restless persons like to have something with which to occupy their hands, and so long as they are allowed to do these things they remain comparatively quiet. Other activities requiring more attention with constant change of direction stimulate and increase the patient's interest. By these methods one would apply quieting or stimulating methods which were effective and should be used more commonly.

Third, activities which he considered even more important were contained in the "educational" group. In thinking of one's physiology one realizes that the whole purpose of the nervous mechanism is to do something. The situation here in regard to the occupational methods was much the same as that in the children's school. The child does not go to school, at least in the primary grades, with the idea of acquiring certain information. It does not matter, for example, whether he learns to read or write, for that will come later if he learns to apply himself steadily. Similarly in occupational therapy, it is unimportant what the patient does. The main purpose is to train him to apply himself to some job steadily in such a way as will enable him afterward to meet situations in life.

Dr. Singer expressed strongly the feeling that most important in occupational work are not the articles which the patient turns out. Nothing does more harm than such a view. The real product is the education acquired by the patient. It matters not what the patient does so long as he is trained along lines of steady application, which is essential to social adjustment. The educational side of occupational work is directed toward developing this faculty. At first the occupation must be selected so as to be interesting and attractive to the individual and from that lead up to things not so attractive but more useful for a vocation.

Thence it is important in the early stage to use for occupation activities which are essentially appealing, to make use of bright colors, music and things pleasing to the senses.

In state hospitals it used to be the practice to grade patients according to their disabilities—the restless ward, untidy ward, and so on. Now they are attempting to group patients according to what they can do rather than what they cannot do. Nevertheless, the disabilities must be considered; for example, some place must be provided for the very restless patients where they can be put under more or less symptomatic treatment. But where education becomes possible, the subdivision can be made according to the kind of treatment needed. Thus, some patients' attention is so low that they must be taught the simplest things. Instead of putting them, as used to be done, where they may be untidy if they wish, they are placed where they will be trained to be tidy. The various grades of occupation used for treatment above this simple habit training are selected for the degree of attention which is required to carry them out. As the capacity for attention develops, the patient should be promoted to occupations of increasing complexity.

Finally comes an effort to train the patient for some vocation, either inside or outside the hospital. If occupational therapy corresponds to primary education this feature can be compared with secondary education. Here the actual produce of the patient's activity becomes increasingly important.

Book Reviews

DIE SYPHILIS DES ZENTRALNERVENSYSTEMS; IHRE URSACHEN UND BEHANDLUNG (SYPHILIS OF THE CENTRAL NERVOUS SYSTEM; ITS PATHOGENESIS AND TREATMENT). By WILHELM GENNERICH. Pp. 265. Berlin: Julius Springer, 1921.

Gennerich has produced a monograph on syphilis of the central nervous system considered from the point of view of the syphilologist who looks on central nervous system involvement as merely one incident in the course of syphilis. His is not the point of view of the neurologist; he is not interested especially in symptomatology or nosology or even to any large extent in pathology. What he has done is to attempt to study the life history (Ursachen) of syphilis as it affects the central nervous system from the primary stage until the end of life of the individual. This study is made on the basis of his own tremendous personal experience and deals especially with the way in which the disease may be modified by treatment. It will be recalled that Gennerich has long been identified with the subject of neurorecidive and has entered into the controversy concerning the production of neurorecidive by arsphenamin. We may therefore expect to find him devoting considerable attention in his book to this subject.

A further interest has been the early involvement of the central nervous system, and Gennerich has been one of those who has contributed in large part to our knowledge concerning the high frequency with which the spirochete makes its entrance into the central nervous system during the early stages of the disorder. The book, then, may be said to have a definite philosophy; namely, that of studying the involvement of the central nervous system in all cases of syphilis in an attempt to understand why some cases have serious involvement while others escape with very little clinical symptomatology, and of observing how the various types of treatment affect its course.

The reviewer believes that the book is the most important contribution of its type that has been made. Probably no one else has had the wealth of material to draw on or has kept as satisfactory control over cases for so long a period of time. What is especially pertinent is that he has made a complete, if not universal, study of the cerebrospinal fluid of his cases of syphilis. Before discussing his theories and his contribution in detail one must draw attention to certain therapeutic points peculiar to Gennerich. It was in the summer of 1916 that he first became thoroughly interested in the intraspinal treatment and from that time on he became its most ardent champion. His intraspinal technic is that of the French, namely, the introduction of neoarsphenamin directly into the cerebrospinal fluid, which is then reinjected into the spinal canal. He has been using a routine type of general treatment consisting of a series of eight arsphenamin injections followed by a series of intramuscular injections of mercury. Thus, his whole general treatment of syphilis, whether the nervous system is involved or not, is practically the same until he adopts intraspinal injections. We, therefore, must keep in mind at all times that his conclusions are based on a rather stringent routine which does not, apparently, vary to any large extent from one case to another. He does not use the intensive general treatment or the long continued less intensive injections of arsphenamin so frequently adopted in the United States, nor has he, apparently, had any experience with spinal drainage, intraventricular, or cerebrosubdural injections, Swift-Ellis technic or the mercurialized serum of Byrnes.

Gennerich states that syphilis of the central nervous system always begins with an involvement of the meninges. In all cases of early syphilis there is "infection of the cerebrospinal fluid," that is, the spirochete makes its way into this fluid. Only a limited number, however, develop clinical symptoms that are discernible. The resultant effect on the individual depends on two things—the immunity reaction of the host and the effect of specific treatment. This immunity reaction as produced by the individual, is, to his mind, the most important concept of all. On this will depend the latter course of the disease, and treatment must be considered from two standpoints, first, what it does to the organism, and second, what it does to the immunity reaction which would normally occur.

Treatment, irrespective of immunity effects, is going to be valuable for the central nervous system in so far as a drug can make its entrance into it. This, according to Gennerich, will depend on the condition of the meninges. He states that the peculiar anatomy and physiology of the pia is of primary importance and that as, in his opinion, the pial surface is nourished by the blood supply only on one side, the effect of such a drug as arsenic is not so satisfactory here as in tissues that have better nourishment. In order to have arsphenamin reach the nervous system it must be in very high concentration in the blood. Illustrating this, he takes up the matter of jaundice, such as one sees frequently after treatment with arsphenamin. He states that one not infrequently finds that the pia and the cerebrospinal fluid become colored, but always at a later period than the tinging of the sclera, and that this coloration of the pia remains for a considerable period after the sclera has again become normal. If it is true that it takes a higher concentration of icteric pigment to enter the cerebrospinal fluid and pia, then a high concentration would be even more necessary for a substance like arsphenamin. The condition of the pia determines the amount of diffusion of the drug to a most important extent. Where the pia has been involved by the disease process and has become thickened and changed, the possibility of the diffusion of the drug is greatly lessened. Thus, the cases in which the pia has undergone chronic changes offer the poorest chances for therapeutic results through intravenous treatment.

Metasyphilis is a term which he thinks should be retained to represent the cases of paresis, tabes and the like, and he would classify metasyphilis as a late form of secondary syphilis stating that all cases of metasyphilis begin in the secondary period of the disease. The determining factor in the development of the more benign central nervous system syphilis or the more malignant metasyphilis is the hydrostatic pressure of the cerebrospinal fluid. Where this becomes increased, leading to the diffusion of the organisms into the deep-lying spaces of the nervous system and associated with a considerable pressure degeneration caused by the high pressure, metasyphilis results. These hypotheses are purely logical deductions, and Gennerich gives no physiologic or pathologic data to maintain them. He merely states that they are so. As working hypotheses they may be useful, as they underly his therapeutic attempts and his therapeutic reasoning. It may be said at once that Gennerich is in no way afflicted with a folie de doute; he apparently enjoys complete and thorough belief in his own dogma. These theoretical deductions are the weak points in the development of his thesis and his book. Fortunately, however, they are only a small part of what he has produced. His work is very completely illustrated by case histories exemplifying and amplifying his statements, and it is on the presentation and grouping of his vast and well controlled material that one must concern oneself in studying his monograph.

He makes his entrance into the study of the subject by considering the results of the treatment of syphilis by mercury and iodids in the pre-arsphenamin days. His general conclusion is that mercury plays very little part in influencing the life process of syphilis in so far as the involvement of the central nervous system is concerned. He finds that there is no great difference in the central nervous system involvement of the patients who had received thorough treatment with mercury and those who had received none. The most that is accomplished is to lengthen the time before the serious damage becomes apparent. However this idea may strike one, it must be said that Gennerich has reviewed the cases of patients treated by mercurials in a most satisfactory fashion, and the conclusions obtained are based on the results. He finds that the results of spinal fluid examinations in patients who had received the classic course of mercurial treatment are far from being as satisfactory as has been generally believed. This work, it seems to the reviewer, is of the greatest importance and must be accepted or dismissed on the results of further study of similar material. Until we do reach a satisfactory conclusion to this matter we have no good basis for an understanding of mercury as an agent in the therapy of syphilis. It may be pointed out, however, at this time that Gennerich's point of attack is not that of the neurologist. Despite the fact that he gives evidence throughout of knowing the difference between the so-called meningo-neurorecidive without clinical symptoms and the more important manifestations which are shown by clinical symptoms, yet throughout he appears to overstress the spinal fluid findings. Thus, in regard to his conclusion concerning the inefficacy of mercury one assumes that he considers abnormal spinal fluid findings of equal importance with actual clinical manifestations. Throughout he seems to indicate that a continued abnormal spinal fluid is merely an early stage of tabes or paresis, but he does not consider the possibility of a person continuing to the end of his days with a positive spinal fluid without any serious mishaps.

Gennerich divides his cases of early syphilis with meningeal involvement into three groups according to the therapeutic results obtained. The first group, representing the vast majority of cases, is made up of those in which the meningeal involvement clears up rapidly with general treatment. The second is a group of cases in which general treatment fails to affect the meningeal process. The cases of the second group will arise despite good intensive general treatment. This raises the comprehensive question of arsphenamin neurorecidives. Is this meningeal involvement the result of irritation by arsphenamin? Gennerich says decidedly that this is not the case, and his chief proof is that these patients recover when treated by intraspinal therapy. All cases, then, of this second group of Gennerich, although developing in the presence of satisfactory general treatment, nevertheless entirely recover when intraspinal injections are given. The third group of cases is composed of those in which the meningeal involvement arises on the basis of a poorly carried out general treatment. These patients are also generally cured by intraspinal treatment. The available space does not permit a complete review of this matter as taken up by Gennerich, but he illustrates very well these three groups with case histories. One wonders whether more intensive general treatment might not accomplish more than was accomplished by Gennerich, but certainly his division of cases is interesting and valuable.

One of the most interesting of the theoretical points is that of the immunity reaction mentioned in the foregoing. According to the author, when the spirochete enters the body it is met by a certain protective mechanism of the patient. This resistance of the body is shown by the generalized symptoms, such as the skin reactions. When these are florid, he believes that the patient is putting up a strong resistance. When the skin reactions are absent the resistance is much less, hence these patients later develop serious disorders such as tabes, paresis, aneurysm and the like. When treatment is given the chemicals tend to check the activity of the infecting organism, but in so doing they tend to cut down the amount of resistance offered by the host. When the treatment is inadequate, that is, when the disease is not cured by killing the spirochetes completely, one is left with a situation in which the resistance of the host is much diminished, whereas organisms are still present in the body and have a chance to develop later and cause serious difficulties. Mercury does not cause such rapid destruction of the spirochete as does arsphenamin, hence the immunity is not diminished to such an extent. He believes that unless treatment with arsphenamin is given vigorously, the results may be worse than if it were not given at all. His statistics, particularly in regard to central nervous system involvement, are exceedingly fertile in showing the effects of smaller and larger amounts of arsphenamin and mercury in the treatment of early syphilis.

Gennerich divides his book into chapters, taking up the treatment of the various forms of syphilis of the nervous system including basal meningitis, meningitis of the convexity, syphilitic epilepsy, tabes, myelitis, paresis and the like. His general thesis concerning treatment is usually simple, namely, that those patients in whom the meninges are quite intact will react well to general treatment. All other patients, and this includes the major portion of patients with symptoms, require intraspinal therapy. By this means he believes the arsphenamin reaches the point of injury.

In reading his text and his cases one is struck by his great optimism as to the results obtained when this method is utilized, and one does not see that he changes his technic to any extent whatever the type of nervous system syphilis with which he deals. Whatever one's point of view concerning the value of intraspinal therapy in the treatment of syphilis of the central nervous system, one is impressed by the results which he reports. His case histories are startlingly interesting. In case after case he shows the development of symptoms and an abnormal spinal fluid despite repeated courses of general treatment, while the same patients improve clinically and serologically to such a degree that they are quite normal after a small amount of intraspinal therapy. One must accept his findings to a certain extent, no matter what one's preconceived ideas may have been. At any rate, it is necessary to look over one's own cases in comparison with his results. He reports the results of treatment under each type of involvement and localization. He maintains that whereas in many cases in which there is a chronic nervous system involvement, symptomatic improvement may occur by the use of general treatment, still these cases will result in paresis or tabes unless the intraspinal treatment is used, in which circumstance he believes the later course will be quite satisfactory. It is feared that his pessimism about the use of general treatment and his optimism concerning intraspinal treatment are both a little excessive being based on a prejudice rather than on a careful sifting of results

He claims excellent and satisfactory results in the treatment of tabes, including cases of optic atrophy and gastric crises. While he does not maintain that the symptoms can be entirely alleviated, he still states that much improvement can result and the progress be entirely halted. He is also optimistic as to the results of treatment in general paresis. He says that almost every case can be greatly helped, and long remissions of four, five or more years can be obtained in a number of the cases. That more satisfactory results equivalent to a complete cure cannot be obtained in the majority of the cases of paresis is due rather to the damage produced by the hydrostatic pressure of the cerebrospinal fluid than to the direct action of the spirochete. He believes that the activity of the spirochete can be entirely controlled in these cases by the use of intraspinal therapy, but because we cannot modify the pressure changes that have developed and which will continue to develop the results are ultimately unsatisfactory.

One cannot study this work of Gennerich without finding many points for criticism, but, on the other hand, one cannot help but draw the utmost inspiration from his work. The reviewer believes that this is the most thorough study of syphilis of the central nervous system that has yet been offered. It is an attempt really to understand what happens to the patient and what can be accomplished by treatment. Certainly such a study opens the way for a much better understanding of the effects of syphilis on the nervous system than can be obtained in any other way. Many befogged ideas have been thoroughly dispelled by his study of a large number of cases showing what really happens. It seems to us that this is a most monumental contribution and that every syphilologist and neurologist owes it to himself to give this work the most careful study.

KURSUS DER PSYCHOTHERAPIE UND DES HYPNOTISMUS (COURSE ON PSYCHOTHERAPY AND HYPNOTISM). By George Flatau. Third Edition. Pp. 175. Berlin: S. Karger, 1920.

The author presents a systematic and detailed review of the subject of psychotherapy. He begins with a brief historical sketch of the treatment of nervous diseases by procedures in which mental factors are the important element. He then proceeds to a somewhat conventional series of psychologic definitions and discussions, after which he takes up the various methods of psychotherapy, devoting the major part of the book to hypnotic procedure.

Dubois treatment and Weir-Mitchell treatment are discussed in a common sense way. In the discussion of the methods of hypnotism the author brings forward no new point of view. He utilizes hypnotism along with the usual common sense hygienic measures, and, although he talks of the hypnotic treatment of masturbation as being "a necessary and often brilliantly efficient means of reinforcement," he furnishes little evidence to show that it is a method which cannot easily be dispensed with.

In the final chapter of the book the author gives a somewhat formal presentation of Freud's psychanalysis with regard to which he is far from enthusiastic.

The book may be recommended to the general practitioner who wishes to know something about the general methods of psychotherapy that are in vogue, but brings little that is new or suggestive to the worker in this special field.

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A CASE OF SLEEP LASTING FIVE YEARS, WITH LOSS OF SENSE OF REALITY *

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At a meeting of the Société de Neurologie de Paris, held the beginning of May, I was delegated to present the cordial greetings of that society to the American Neurological Association. I assure you that it affords me much pleasure to be the bearer of such greetings and to have the opportunity of expressing to you my appreciation of the hearty welcome that you have given me. I also wish to thank you for the interest that you have shown in my studies in pathologic psychology. Disorders of the psyche play an important rôle in nervous diseases and to understand such disorders often is essential. Hence I am led to present to you this study of the remarkable case of a young woman, aged 23, whom I was able to observe for several years at the Salpêtrière: a case of abnormal sleep prolonged for more than five years and of that strange disorder of apperception which seems to rob things of their reality, a disorder which presents many interesting problems.

HISTORY OF THE CASE

There is little to be said of the heredity of the patient. A grandmother, whom the patient resembles, was nervous and easily agitated. However, this young woman had always been quite different from her brothers and sisters and had been regarded as markedly neuropathic. She was unstable; sometimes very gay and exalted, at other times seclusive and gloomy. Complaining continually of extreme weariness, which came on rapidly, she worked very irregularly, although possessed of marked ability and rather unusual intelligence. Menstruation began at the age of 16, and has always been very irregular. The occupation she was to learn fatigued and distressed her, and at about the age of 13 she began to have paroxysmal disorders of consciousness in the form of fainting spells more or less prolonged.

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* Read at the Forty-Seventh Annual Meeting of the American Neurological Association, Atlantic City, N. J., June, 1921.

The first bad attack occurred at the age ot 14 during a religious ceremony and was explained by the patient as follows: "This feeling always takes me when I am placed in a difficult situation—when the illumination is too brilliant or there is too big a crowd; or when I must do anything that is fatiguing or enervating. In a few minutes I am gone; I do not know what I am doing; I act like a somnambulist, an automaton; I do things without knowing that I do them, and no doubt I do stupid things. It is not I who order my movements; it is no longer I who act and speak." When the attacks were especially severe, she would gradually become motionless in a chair, close her eyes and apparently fall asleep. One would have to undress her and put her to bed, where for several hours she would remain inert. As time went on, attacks of this sort grew more and more frequent; they also were of longer duration, and at the age of 17 she began to sleep continuously.

CONDITION OF PATIENT ON ADMISSION TO HOSPITAL

She was admitted to the Salpêtrière, where for the last four years I have been observing her under favorable conditions. When brought to the hospital the patient was apparently sound asleep, and she remained in this condition almost without change for the first year. With eyes closed she scarcely stirred in bed, made no reply to questions and reacted but slightly if handled or pinched. The noise made by other patients did not seem to interest her in the slightest. During the bombardments of the war she gave no evidence of being disturbed, even when a shell burst near by. She asked for nothing, and did not open her mouth when offered food. However, she was easily fed without a tube. When the nostrils were pinched shut she opened her mouth a trifle and food placed in her mouth was promptly swallowed. She was badly constipated, there being no movement except by enema every second day. The urine was quietly passed in bed. Menstruation occurred only every three or four months. From time to time, especially during menstruation, sleep was disturbed by convulsive attacks, with large movements, during which the patient was almost always thrown out of bed. During such spells the patient spoke not a word and showed no signs of delirium. The convulsion lasted a few minutes, after which she slept as calmly as before.

Under these conditions one might easily believe that psychologic processes were reduced to the minimum—which would be a serious error. The parents of the patient had already informed me that it was well to leave at her bedside cakes, fruit and other food. Sometimes, during the night, when she thought she was not observed, she would stretch out her hand, take some of the food and eat a part of it.

EXPERIMENTS IN AROUSING THE PATIENT

I soon found that if I persistently spoke to her I could elicit a faint smile, accompanied by slight movements of the hand. I then began to talk to her just as if she could understand all I said. I explained to her that I wished to mesmerize her in order to get her into a different somnambulant state in which she would be able to reply to my questions. This conversation, which was accompanied by mesmeric passes, or simulated passes, gradually brought about a change in the patient's attitude. Little by little she was brought to open her eyes, sit up in bed and to talk with me in the most intelligent way. I thus secured about once a week a talk of from a quarter to a half hour, after which time the patient seemed fatigued, replied with greater effort and then dismissed me with an odd expression, to be explained presently: "Why do you want me to talk to you? You do not exist, neither do I. Good night." And she would lie back on the bed and fall into slumber lasting a week.

During these supposed hypnotic séances and the short conversations that I was thus able to have with her, I succeeded in getting her to promise that from time to time, when she could, in the same manner as she took the cakes during the night, she would take the pencil and paper that I placed within her reach and write me a letter. This "game" seemed to please her, as before long she got into the habit of rousing on certain nights when she thought no one was looking and of composing for me long rambling notes, which she then carefully concealed in a hole in the mattress lest they fall into the hands of any one but myself. Things went on thus during the first two years. Her prolonged sleep was interrupted by these short conversations about once a week, and two or three times a month she would write me a letter. Although these interruptions were very short, they gave me an insight into the peculiar mental state which accompanied her prolonged slumber.

PHYSICAL AND MENTAL CONDITION

First, it is to be noted that motion, sensation and elementary psychic processes were entirely intact, except that possibly during the convulsive seizures there was some blunting of sensation and less force of the movements on the right side. The reflexes, which were repeatedly examined and further investigated by Dr. Souques, were entirely normal. From time to time, cramps or contractions of the abdominal muscles were observed, but were easily dispelled by a little massage. There was some retraction of the right foot, apparently caused by the weight of the bed clothes, extending over a long period of time, and antedating her admission to the hospital. In short, there were no signs of organic disease of the nervous system. Evidently we had to do with a psychopathic disorder.

In investigating the mental state of the patient we must not be led astray by the first impressions she tried to convey by her conversation and letters. She insisted that she was in a state of frightful delirium—of profound dementia: "I spend all my time," she said, "in a horrible state of wandering. Pay no attention to a single word I say. I don't know where I am, I understand nothing of what is going on, remember absolutely nothing. The crazy people around me are far more sensible than I. At least they know what they are dreaming about. I am lower than the worst insanity. I am a living corpse. For me all is unconsciousness and irrationality in the midst of darkness and nothingness." This exaggeration and this pathos put us on our guard; we easily recognized "ce jeu des combles" of the hyperconscientious or phobic patient.[1] It was easy to see that she had neither the dementia nor the delirium which she imagined. On the contrary, she exercised her intellectual faculties in a most interesting way.

In spite of her assertions to the contrary, she knew perfectly where she was and repeatedly made very apt and amusing comments on members of the personnel and on the other patients, whom she called her worshippers because they came to gaze with wonder at the persistent sleeper. She laughingly told about another patient taking her hand and telling her fortune from the lines of the palm. She complained of a nurse coming at night and taking chocolates from the bag that her relatives had left on the stand by the bedside. One day she even requested me to ask her parents to send her some articles for a layette as she had noticed that one of the nurses was pregnant. The same keenness applied to recollections of her early life. In her letters she recounted amusing details from every period of her life.

Although she had received only an elementary education, she showed remarkable intelligence and a lively imagination. In the papers that she wrote for me were all sorts of philosophic ideas and endless discussions of metaphysical problems, which seemed intensely to interest her. As she said herself, these speculations attracted and at the same time were unwholesome for her. It was often after meditations of this sort that formerly she had had the disorders of consciousness and fainting spells. She also wrote little romances, puerile in conception but rich in imagination. She showed a peculiar taste for poetry and gave me many verses which she said she had written. Persons to whom I showed them thought they recognized something familiar, and very likely this was true. Probably she more or less consciously mixed memories with her own compositions. None the less, one received the impression that here was an unusually active intelligence, which appeared all the more strange in view of her prolonged sleep and apparent incontinence. When reminded that in writing all these things she couldn't be so stupid, she angrily replied: "One may be an idiot for real life but intellectually not one at all. Don't you know that one may be a fool in some things and not in others?"

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[1] Obsessions et psychastenie **1**:61, 63, 1903.

THE SENSE OF UNREALITY

It is, indeed, in what she called real life, or rather in the sense of the reality of life, that we shall find very interesting psychologic disturbances. First, let us mention the feeling of automatism; "For a long time," she averred, "I have been acting only by reason of some incomprehensible instinct without knowing what I do . . . My will counts for nought . . . The person who makes me act must be outside of me. I speak without willing it, and if I don't talk foolishness it's no fault of mine. . . . With astonishment I hear myself talking," . . . etc.

The most interesting sensation is the feeling of unreality in all its forms: "I don't see things as I used to. . . . I see things devoid of substance. . . . They must be hallucinations and not real objects. . . . Things act only on my eyes, not on my brain. Doubtless I see everything, without doubt nothing is changed, except that things are not real. . . . It reminds me of the time when I had to take castor oil and some one would pinch my nose so I should not get the odor. I perceive things, but I have no sense of their taste." She described numerous occasions on which the feeling of unreality took possession of her: "When I did something too quickly, when I ran anywhere, I would suddenly stop, quite astonished, for my indolent brain had not been as quick as my legs; it had remained at the starting point and I was surprised to find that I had arrived. I had fallen from the moon and things no longer existed as they had. True, the appearance of things is just the same. I could describe them to you just as you would yourself, but what I see is only a play, a Punch and Judy show; it is clumsy, vulgar, unpleasant and, above all, false; it doesn't really exist."

She not only had doubts as to the existence of objects but also as to their character; for example, she would look at her mother and say to herself that this woman (whom she recognized full well) was not her mother, further, that this woman did not really exist. But it was especially on her own personality that her doubts were most keenly focused or concentrated. "I do not know how I live or whether I live at all; I am a body without a soul or a soul without a body, whichever you like; I cannot be dead, for I feel my heart beating, but is this heart mine and who am I? I must be a mechanical animal, the latest creation of science. But an automaton does not think. How is it that I can think?" Without suspecting the enormity of the proposition, which would horrify the shades of Descartes, she always concluded: "At any rate, I think—but I do not exist."

It would take a long report, such as I hope to make later, to present all the forms that the feeling of unreality assumed in the case of this patient and all the statements that she made on the subject. But I wish to emphasize one feature that I have rarely observed so fully developed: the feeling of unreality as regards one's recollections. This patient, who related in minute detail numerous events of her past life, constantly interrupted herself to insist that she had no memory at all: "I know full well that I do not actually remember . . . I know it wasn't I who experienced all that. . . . These are not things that really happened to me. . . . I am relating all that, but it seems to me that I might just as well recount something else. Perhaps it is a story that I have read, or a tale that I am inventing. . . . It seems to me that I have had no past existence, and yet I know a lot of things about this individual whom you call by my name—also things that others do not know. It is harder to understand than the mystery of the Holy Trinity."

It was this feeling of the unreality of things, persons and memories which formerly, in the beginning of her trouble, suddenly overwhelmed the patient in the midst of some activity and brought on the disorders that she was unable to explain. At first this feeling quickly disappeared after a little time; then it became more prolonged and finally continuous. It was this perpetual doubt, this feeling of unreality in regard to all objects, all persons, her own self and the recollection of all past events that constituted the essential mental disorder underlying this peculiar sleep lasting several years.

INTERPRETATION OF SYNDROME OF UNREALITY

While agreeing with the patient that the whole thing is very complex, we know that this syndrome of unreality is rather well known and has been variously interpreted. Although I cannot now enter into a discussion of these theories, I may remark that the present case again confirms the interpretation which I proposed long ago.[2] As I have repeatedly stated, it does not seem possible to ascribe this sense of unreality to modifications of the elementary sensations or perceptions, even though one has in mind more particularly muscular or visceral sensations (myopsychic or somatopsychic). Like all such patients, she presented absolutely no disorder of the elementary sensations. All muscular and visceral sensations were normal. One should not try to explain an observed symptom by suppositious symptoms assumed for the needs of argument. I am always inclined to believe that there is a close relation between disorders of the sense of reality and disorders of volitional activity, which in this patient were predominant from the start. In her case we find all sorts of abulia, timidity, indecision, doubt, disinterest, impulsiveness, need of being directed by others, etc. All these disorders followed performance of the slightest task or any prolonged slight effort. Even at the present time, when she appears to be wide awake, I need only direct her attention a little more closely to what I am saying and force her to concentrate an instant, to precipitate a flood of doubt or anxiety or a convulsive attack and to cause her to fall again into deep slumber. While in this brief summary of the case I cannot enter upon a discussion of the point, I am inclined to think that the sense of unreality does not depend on impairment of elementary sensations or elementary psychologic phenomena, for I find no such impairment, but that it depends rather on a reduction of activity, a lack of interest in activity and especially on disturbance of deliberate action, which is, indeed, the very starting point of what we call reality.

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[2] Névroses et idées fixes **1**:40. Footnote 1, pp. 282, 299. La tens in psychologique ses degrés, ses oscillation, Brit. J. Psychol., Medical Section, July, 1821, p. 214.

Reflection or deliberation is a higher form of integration of behavior; it is an integration of ideas instead of reflexes; it effects a synthesis of the various tendencies awakened, combines them into a definite line of conduct which gives us unity and permanence. For us, individuals are real in proportion to our awareness that we love them or hate them; it is the emotional notion or concept that makes the real. Deliberative action, when it can exist, transforms perceptions. It is not sufficiently recognized that perceptions vary with the different levels of psychologic tension. At the level of reflection the tendencies awakened by a known object are at one and the same time numerous and unified. We are conscious of a stable attitude toward every such object. The same is true of our memories at the level of reflection. We know that we shall always affirm them in the same way. On the other hand, she is never sure of her attitude toward the object that she sees and about which she endeavors to reflect; she doesn't know whether the reminiscence affirmed now will be reaffirmed later. She even indulgently informs one that the very next minute she may assert just the opposite. It is this weakness, this ready exhaustion of her reflective power, that causes her perpetual doubt and her sense of the unreality.

FURTHER CONCLUSIONS

All this may seem far indeed from the slumber of our patient, a sleep which has lasted for five years and which seems to be the essential feature of her trouble. Of course she sleeps, and sometimes the sleep is more profound and more prolonged than normal. But I do not believe that her state during the last five years has been constantly one of sleep. This young woman appears to sleep much more than she really does. Does she then, pretend to sleep? One may perhaps, call it simulation, if at the same time we concede that the young woman does not in the least prevaricate. She deceives herself and is the first one to believe in her sleep. It is a case of irrational conviction, which is frequent among psychasthenics, in whom the feeling of inadequacy and the obsessions frequently engender delirious states whenever psychic activities are depressed below the level of reflection. Before the Psychopathological Association in Boston, I presented the interesting case of a psychasthenic with obsessions who, from time to time, developed a delirious state characterized by crude or elementary affirmations. I am inclined to believe that these delusions of inactivity, of fatigue, of hallucination, of dementia and of sleep, play a more significant rôle than is generally recognized.

This patient is also a psychasthenic with obsessions, devoid of will power and of the power of reflection. She doubts everything and suffers from a feeling of unreality, at first momentary, later permanent. She gradually became detached from real life and lost interest in it, and that just when she had a fear of the future—fear of life itself. "Life is all a hoax," she said. Little by little she took refuge in delusions and in sleep, which first occurred in transitory fashion during her attacks of depression. When the depression became more profound she began to believe in her continuous sleep. As she doubted everything, she acquired the delusional conviction of her sleep and insanity. This it was that has held her in the pose of continuous sleep for five years.

If these conclusions are correct, this disorder is curable, for states of depression at this level rarely are permanent. By means of the stimuli already noted, I have induced the patient to speak and to do more and more. During the last year, progress has been particularly marked. First, her sleep became less profound, it was easier to awaken her and to keep her clean, to get her to feed herself, to dress herself, and to walk about. For the last six months she has been living with her own family. She is no longer actually somnolent except during the forenoon. During the afternoon she has developed considerable activity, not quite normal of course, but nearly so. The feeling of unreality has remained about the same. This was, I think, the fundamental symptom and is therefore the most persistent.

This curious case of psychasthenic depression with doubt and fear of life, producing a delusion of sleep and the appearance of sleep for five years, shows us, as I have said, how interesting psychologic interpretation may be in nervous disorders.

ABSTRACT OF DISCUSSION

Dr. Hugh T. Patrick of Chicago said that apparently Professor Janet had stopped at a point at which the patient believed that she was asleep and did not exist. Evidently that was his answer to the question why she apparently was asleep and why she acted as if she had lost reality. Dr. Patrick believed there was one additional step to take. Why did she finally believe that she was asleep, and why did she finally believe that she had no existence? It was because she wished to be asleep, and because she wished to have no existence. This obviously bore also on the prognosis, the curability and the therapeutics. In other words, such a patient would promptly and reasonably get well when conditions were such that she ardently wished to be alive, to exist, and wanted to be awake.

The therapeutic measure, then, should be to bring about such conditions that the reaction would not be this defense reaction of creeping into a hole of unconsciousness, but that the patient would wish to come out into the world as it is and partake of the general troubles which we all more or less enjoy.

Dr. Foster Kennedy of New York asked Dr. Janet whether he did not consider hysteria invariably as a refuge, as a temporary and inadequate solution of a problem to which the organism could not properly react or solve.

In olden times our forefathers would retreat to a marsh or desert, there to escape the woes and troubles of a tempestuous world; later they saved themselves in monasteries from having to adapt themselves to a difficult environment, and he thought perhaps Dr. Janet's patient had effected a modern solution of an ancient problem.

Professor Janet of Paris stated, in answer to the question as to whether there were during the years of loss of sense of reality any periods of what might be called real sleep, that on several occasions the patient exhibited periods of profound sleep, which was deeper than normal sleep.

In regard to Dr. Patrick's question, Dr. Janet felt that such patients were really incapable of normally desiring anything—either the desire of getting better or the desire of taking refuge into a state of sleep or nonexistence. He felt this was a sort of automatic refuge rather than one that was desired.

THE SUCCESSFUL REMOVAL OF BRAIN TUMORS

REPORT OF A SERIES OF EIGHT CASES *

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In presenting for review this series of tumors of the brain, successfully removed, we have selected tumors of various types and various localities as representing different problems in localization and in surgical management. In consideration of the operability of brain tumors, the reports in current literature are frequently misleading. For example, many cases are recorded as brain tumors in which the brain tumor was not seen at operation or verified at necropsy. For practical considerations such reports are of no value as neither the nature of the tumor nor its precise location is specified. It cannot be classified on the basis of its pathology nor with relation to accessibility since its precise location remains undetermined. These two phases of brain tumor, character and location, are the only ones of practical moment when considering the question of operability.

Then, too, in estimating the incidence of operable in contrast with inoperable tumors, the time has come when a classification should be made and adopted as a basis of all discussions. It is quite as improper to include all kinds of tumors of the brain in one group when attempting to evaluate operative possibilities or operative risks as it would be to discuss under one heading all surgical lesions of the stomach. There is as much difference between the sharply defined endothelioma and the subcortical infiltrating glioma as there is between sharply defined gastric ulcer and the diffuse carcinoma. In the stomach the latter represent separate and distinct problems; quite as separate and distinct are the problems presented by various tumor types of the brain.

The following classification might be acceptable.

Pretentorial
(a) Benign Tumors
    1. Fibroma
    2. Osteoma
    3. Enchondroma
    4. Tuberculoma
    5. Endothelioma
    6. Miscellaneous

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* Read at the Forty-Seventh Annual Meeting of the American Neurological Association, Atlantic City, N. J., June, 1921.

(b) Malignant Tumors
    1. Glioma
        (a) Cortical, subcortical
        (b) Infiltrating, circumscribed.
    2. Endothelioma
    3. Tuberculoma
    4. Miscellaneous
(c) Pituitary Lesions
(d) Miscellaneous
Subtentorial
    1. Acoustic Tumors
    2. Angle Tumors (Not Acoustic)
    3. Hemispheric Growths
        (a) Endothelioma
        (b) Fibroma
        (c) Glioma
        (d) Tuberculoma
        (d) Miscellaneous

This classification would afford a basis for intelligent discussion and for the comparative studies of reports from various sources. It is manifestly unfair and misleading in considering the question of operative risk to consider tumors of all types together. For example, operations even though only exploratory, in cases of subcortical glioma are always hazardous; and the general mortality of surgical efforts in brain tumor should be reckoned for each of the several groups as proposed in our classification.

Not until, as a class, operations are undertaken at an earlier period will there be any striking change in the incidence of successfully extirpated growths. In a certain number there is an extraordinarily long latent period even though the tumors are not situated in the silent areas of the brain, but in too many instances has there been an unconscionable delay in recognizing tumor probabilities and recommending surgical intervention.

The following cases have been selected as representing tumor types and various locations:

1. An endothelioma of the left occipital lobe
2. A tuberculoma of the parietal lobe near the motor cortex
3. An encapsulated glioma of the motor area
4. A calcified endothelioma of the falx
5. An unusually small endothelioma near the motor cortex
6. A fibroma of the posterior fossa origin undetermined
7. A glioma of the cerebellar hemisphere
8. An acoustic tumor (fibroma).

Two of these cases have clinical features of unusual interest. Case 2 has been employed for a discussion of epilepsia partialis continua affecting the face, and its relation to convulsive movements of the face from irritation of the facial nerve. Case 5 is of special interest because of the small size of the tumor and the importance of aphasia as a localizing symptom. This was a case in which removal of the tumor effected a complete disappearance of all symptoms, a cure in every sense of the word. There has been so much dispute regarding the importance of Broca's area in motor aphasia that Case 5 with sharply defined attacks of motor aphasia and the discovery and removal of a small endothelioma from or very near Broca's area with complete disappearance of these attacks of aphasia is of importance in the controversy regarding aphasia.

REPORT OF CASES

CASE 1.—*Summary: An endothelioma of the occipital lobe removed from a patient, who continued free from recurrence three and a half years after operation.*

History.—M. P., aged 41, was sent to the neurosurgical service of the University Hospital by Dr. R. S. Hooker, as a case of brain tumor. Her previous medical, family and social history contains nothing that bears directly or indirectly on the lesion under consideration.

Four months ago the patient began to complain of frontal headaches, at first observed in the morning, of a few hours' duration. Gradually they increased in severity and continued longer until at the present time they are practically constant. Almost contemporaneous with the headaches dizzy spells developed. These came and went with the headaches. On only one occasion was there complete loss of consciousness and then only for a few moments. With the headaches and dizziness the patient noticed that she was having difficulty in "walking straight" in the dark and more particularly in keeping her equilibrium when going down stairs. Later even in daylight hours she could not walk without staggering. There had never been, so far as she knew, any weakness of either the upper or lower extremities; there had been for several months an aching pain in the legs, especially at night, and edema of the legs coincident with dyspnea and palpitation on exertion. For three months she had been annoyed by vomiting, sometimes once, sometimes three or four times a day, without relation to meals. She thought there had been some diminution in the acuity of her sense of taste, of smell and hearing and some impairment in memory. During the period of her illness she had lost twenty pounds.

Physical Examination.—Cranial Nerves: None of the cranial nerves were involved with the exception of the optic nerve. (See ophthalmic report.)

Motor Function: There was no impairment of movement or strength in either of the upper or lower extremities.

Sensory Function: There was no impairment of any form of sensation, no paresthesia, no loss of sense of position or stereognostic perception.

Reflexes: Biceps and triceps reflexes were equal and normal. The right patellar tendon reflex was more prompt and of slightly greater intensity. There was no ankle clonus and no Babinski sign.

Coordination and Equilibrium: There was no ataxia in the upper extremities. Diadokokinesis was good in both hands. A suspicion of asynergia was aroused by the right finger to nose test. Standing with eyes closed the patient swayed slightly, chiefly to the right.

Ophthalmic Report: The pupils reacted sluggishly to light and accommodation. Vision in the right eye was 6/60; papilledema, plus 5 diopters. Vision in the left eye was 6/30; papilledema, plus 4 diopters. There was right lateral homonymous hemianopsia.

Ear Report: Labyrinths, auditory nerves and brain-stem appeared normal. Bárány tests suggested pressure over the cerebellum of supratentorial growth.

Roentgen-Ray Report: The posterior clinoid processes were somewhat attenuated. The sella turcica was slightly enlarged. There was a possible suprasellar growth. The right internal auditory meatus was larger than the left.

Miscellaneous: The Wassermann reaction of the blood and spinal fluid was negative. The spinal pressure was 32 mm. of mercury, and the spinal fluid contained 10 cells. The urinalysis was negative. Hematolysis: hemoglobin, 60 per cent.; white blood cells 9,900; red blood cells 3,420,000.

Summary of Findings: Headache, nausea, vomiting; right lateral homonymous hemianopsia; papilledema; disturbed equilibrium and doubtful asynergia (right).

Operation.—A craniotomy was performed with total extirpation of the tumor. Ether anesthesia was given, endopharyngeal method, in the face down position. A large cranial flap was reflected to uncover the entire left occipital and in part the left parietal lobe. The dura was found to be under tremendous tension. An attempt at puncture of the posterior horn of the ventricle failed. An attempt to evacuate the ventricle by callosal puncture also failed. Simultaneously an incision was made in the dura and a lumbar puncture performed. On withdrawal of a small amount of fluid excessive pressure was controlled. The tumor presented on the mesial surface of the occipital lobe and extended to the base. Differentiation between the brain tissue and tumor was not difficult, and the tumor was enucleated without difficulty. Encapsulation was complete, and there were no attachments except along the surface of the falx. There was moderate bleeding from the falx; this was readily controlled by hot tampons and muscle grafts. The removal of the tumor left a large cavity bounded by the falx, tentorium cerebelli and occipital lobe. The wound was closed (Fig. 1).

Blood Pressure Observations: During the preliminary steps the systolic pressure was 90; on exposure of the tumor the systolic pressure was 80; after removal of the tumor the systolic pressure was 70.

Pathologic Report.—The tumor was well encapsulated and the capsule was intact throughout except at the point of attachment to the falx. The tumor measured 10 cm. in the largest diameter. The histologic diagnosis was endothelioma.

Subsequent History.—The postoperative convalescence was uneventful. The patient was relieved of all her subjective symptoms, her fields now are partially restored, and she is now well and free from recurrence three and a half years after the operation (Figure 1).

Comment.—This case is cited as illustrative of the type of endothelioma growing pretentorial. The rate of growth is invariably slow, and there are well authenticated cases in which the history would imply a period of fourteen years' growth. In this series is included a case in which the symptoms were of ten years' duration, and the tumor must have had its origin a number of years before its existence was suspected. It is interesting in the case we are discussing to note that, in a tumor of that size (diameter, 10 cm.) there had been no subjective or objective evidence of its existence more than four months before its removal. The most plausible explanation for this really sudden onset of symptoms is ventricular distention. There is no doubt that at this time the outlet to the ventricles became obstructed, and for the first time signs of excessive intracranial pressure manifested themselves.

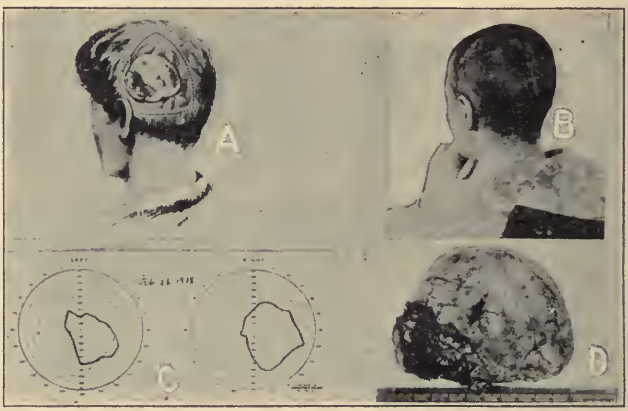

Fig. 1.—*A,* schematic drawing of tumor in situ; *B,* patient two weeks after operation; *C,* right homonymous hemianopsia; *D,* tumor, showing complete encapsulation except at point of origin from falx.

Failure to reach the ventricle on the side of the tumor, either directly or by callosal puncture, would lead one to the conclusion that the ventricle had been compressed and the space practically obliterated by the tumor.

The reason d'etre for some of the symptoms recorded in the history is not clear. Subjectively the patient reported diminution in acuity of sense of taste, of smell and of hearing, but on examination these apparent disturbances were not verified. Taste, smell and hearing were normal.

Had it not been for the homonymous hemianopsia, obviously the characteristic focal symptom, and that at a slightly earlier period the fields were normal, there might have been an error in localization; there was unquestioned evidence of disorder of the right cerebellar hemisphere, the result of the transtentorial pressure of the tumor. It is worthy of note in passing that from the ear clinic the report was returned without any explanatory note that the examination suggested pressure on the cerebellum by a supratentorial lesion. In the words of the ophthalmic report, there was a right homonymous hemianopsia. This is not strictly accurate; the charts portrayed what was really a quadrinopsia. Not much more than a quarter of the fields was involved. It has been said that a quadrinopsia is peculiar to lesions of the temporal lobe. Perhaps this may be an exception to the rule.

There is little to be said by way of comment on the purely surgical aspects of this case. With the localization clearly defined to the occipital lobe the tumor was readily uncovered by the reflection of a flap large enough to include all the occipital and a part of the parietal lobe. In all exposures for lesions posterior to the Rolandic fissure, the patient should be in the prone position on the operating table, that is, with face down. Our extension head-rest designed originally for suboccipital operations, meets all the indications for supra-occipital cases. The encapsulation of the endothelioma facilitates the operator in finding the line of cleavage between the tumor and brain tissue. The problem of hemostasis arises only when separation of the tumor from its area of attachment or point of origin on the falx begins. Sharp hemorrhage is then encountered and may be temporarily controlled by tampons of cotton or, if the area be not too large, by direct pressure with the finger. The latter is preferable as it lends itself to the "postage-stamp" method of control, which originated during the war, for gunshot wounds of the longitudinal sinus. A piece of muscle or aponeurosis, conveniently taken from the temporal muscle, of proper dimensions, is placed on a finger tip of the free hand and, as the finger of the hand controlling the hemorrhage is withdrawn, the finger with the muscle graft takes its place. When hemorrhage was uncontrolled except with the pressure of a large cotton tampon, the tampon was left in place, the wound closed completely and in the course of three or four days reopened and the tampon removed without recurrent bleeding.

The subsequent course of events is as one would anticipate after a radical removal of a tumor of the endothelioma type. There has been no recurrence or signs of recurrence after a period of over three years. Though of larger dimensions than corresponding tumors of the spinal cord, the surgical possibilities closely parallel one another. The endothelioma of the spinal cord and of the brain when pretentorial is distinctly an operable growth in the technical sense of the word and is one of the most favorable tumors for operation. If not removed entirely, however, it may recur and in the recurrence the tumor is not likely to be well defined. An infiltrating endothelioma is distinctly a malignant growth, it is inoperable and of rapid development.

The absence of word deafness and word blindness in a tumor of the left occipital lobe is to be explained by the fact that the tumor was on the mesial side of the cerebral hemisphere, and evidently originated from the falx cerebri to which it was attached. It had its origin from a region of the brain in which the only localizing sign to be expected was hemianopsia. An endothelioma grows very slowly and causes atrophy of the brain proportional to the size of the tumor and therefore may not cause symptoms until it has reached a considerable size unless it is situated in a part of the brain functionally active.

Case 2.—*Summary: The removal of an unusually large tuberculoma from the left hemisphere of an adult, male. The operation performed in two sittings and a ventriculogram made in the interval; recovery.*

History.—U. F., aged 26 years, was referred to the neurosurgical service for operation by Dr. William G. Spiller. There was no history of tuberculosis in the family. The patient had been a noncommissioned officer in the United States army, and had served in France. Before his present illness he was engaged as a hotel porter.

He was perfectly well until nine months before coming under observation. While sitting and talking he said he felt sick and suddenly began to make a grunting noise. His head jerked rapidly back and forth. His eyes were starey, he frothed at the mouth, bit his tongue and voided urine. This attack, in which his legs and arms did not move and he did not fall from the chair, lasted several minutes. He was put on the couch and slept for an hour. For seven months he had similar attacks as often as once a week; he knew when they were coming on and carried a spoon which he put in his mouth so that he would not bite his tongue. Once an attack came on while he was walking to a chair; his head began jerking and, though someone was holding him, there was no tendency to fall. For the past two months the attacks have been mild, and he has not frothed at the mouth or bitten his tongue.

Three months ago he began to have trouble in speaking. He knew what he wanted to say but could not find the word. This irritated him so that he often gave up the attempt to talk. He also stuttered. For the past two months he has been drowsy and continually yawning; he complained of headache which he could not localize; occasionally he has been incontinent both as to bladder and bowel. He has dragged his right foot for some time but not markedly so; he was too weak to lift a chair but could drag it where he wished.

Before his illness he was alert and intelligent and had always done well at work. During the past two months particularly there has been a notable change in his condition.

Physical Examination.—The patient was a well nourished and well developed adult negro. He was stuporous and fell asleep two or three times during the examination. If a question was asked abruptly he answered it, but if followed immediately by a second question there was no response.

Thorax: There was no cardiac enlargement but there was a loud systolic murmur at the apex not transmitted to the axilla. The right side of the chest was slightly more prominent than the left, the sternum inclining to the left. The percussion note was resonant throughout; at the left apex there was prolongation of expiration. There were no râles.

Cranial Nerves: There was bilateral papilledema plus 4 diopters, of the second cranial nerve. There were many retinal hemorrhages. Third, Fourth and Sixth Nerves: The pupils were equal and reacted sluggishly to light. The ocular movements were full in all directions. There was no nystagmus. Seventh Nerve: He did not draw up the right corner of the mouth as well as the left. Frequent rhythmical twitching was observed confined to the right side of the mouth. Twelfth Nerve: Slight deviation of the tongue to the right when protruded was noted. Extremities: The grasp of the right hand was weaker than that of the left. In walking he was uncertain and dragged the right foot.

Reflexes: Biceps and triceps tendon reflexes also patellar tendon reflexes, right and left, were not obtained. The Achilles tendon reflex on the left was absent. The Achilles tendon reflex on the right was very weak.

Dr. Spiller reported: The patient is markedly stuporous but can be aroused and can answer questions correctly. During the examination, he has frequent rythmical twitching confined to the right side of the mouth. This is usually more pronounced when he separates his lips in showing his teeth. He does not draw up the right corner of the mouth as well as the left. It is impossible to test him for hemianopsia because of blindness, but he seems to see the hand in the right fields, but it is impossible to determine this accurately. The tongue deviates a little to the right when protruded. The jerking of the right corner of the mouth is like that of epilepsia partialis continua. He responds to all simple commands and answers in short sentences without any definite aphasic symptoms. He understands all that is said to him, so he is not word deaf. The grasp of the right hand is distinctly weaker than that of the left. The finger to nose test is well performed on both sides. It is impossible to test his sense of position because of his mental condition. In walking he is very uncertain and drags the right foot. I cannot obtain either the patellar reflex or the left Achilles reflex; there is a very weak right Achilles reflex.

Diagnosis: Probably brain tumor of left motor region of cerebrum. It is impossible to localize with accuracy because of the advanced condition of the disorder. The continuous jerking of the right side of his face is suggestive of irritation of the face center of the left motor cortex. It is singular that the right side of the face when the patient is quiet may show no tremor but voluntary movement of mouth, as in showing teeth, at once starts it.

Miscellaneous: The blood contained 4,880,000 red blood cells, 11,800 white blood cells and 90 per cent. hemoglobin. The Wassermann test of the blood was negative. Urinalysis: Specific gravity 1.013, alkaline reaction, mucus + +, no red blood cells, from 5 to 10 white blood cells to the field.

Summary of History and Symptoms: History: (1) convulsive seizures for nine months, confined to the head, involving especially the face; (2) aphasia, three months' duration; (3) headache, drowsiness and incontinence for two months, and (4) weakness of the right arm and leg. Symptoms: (1) frequent rhythmical twitching of the right side of the face; (2) weakness of the right side of the face, upper and lower limbs; (3) both patellar and left Achilles reflex absent, right very weak; (4) headache and drowsiness; (5) papilledema, plus 4 diopters bilateral, and (6) pyuria.

Preoperative Diagnosis: Tumor left motor cortex, near face center.

Operation.—A craniotomy was performed by Dr. Frazier and his staff. At the first sitting ether anesthesia was used, endopharyngeal method. A cranial flap was reflected for exposure of the left motor cortex. When the dura was exposed it was found to be under tremendous pressure. Attempts made to reduce pressure by ventricular puncture on the left side, both direct and by callosal puncture, failed. As it did not seem wise to open the dura until the pressure could be reduced, an attempt was made through separate opening to evacuate the right ventricle. A few cubic centimeters of blood-stained fluid escaped, but the pressure was not affected. At this point the exploration was discontinued and the wound closed.

Ventriculogram.—One week after the first sitting 100 c. c. of fluid were withdrawn intermittently and 100 c.c. of air injected (10 c.c. of fluid withdrawn and 10 c.c. of air injected). The roentgenogram of the skull did not reveal any air shadow in the ventricle. The latter was obliterated evidently by the tumor.

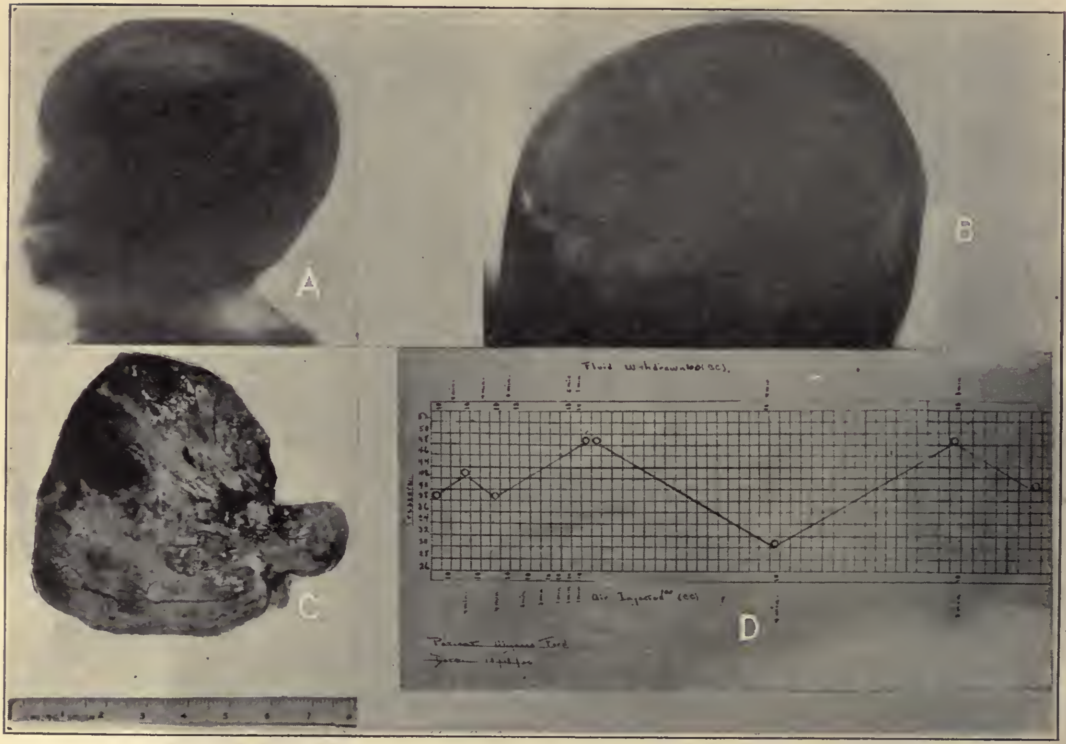

Fig. 2.—*A,* air shadows symmetrical in dilated ventricles of a case of hydrocephalus; *B,* absence of air shadow, ventricles compressed by tumor; *C,* encapsulated tuberculoma which caused collapse of ventricles—photographed immediately after removal; *D,* pressure observations as fluid was withdrawn and air injected.

Exploratory Craniotomy; Second Sitting.—Two weeks after the first sitting and one week after ventriculography exploration was continued. The wound was reopened. The subdural tension was as high as at the first sitting. Lumbar puncture was done with withdrawal of 45 c. c. of cerebrospinal fluid. Immediately tension was restored to normal limits. A dural flap was reflected and the tumor presented at the anterior, inferior quadrant of the opening (near the junction of the Rolandic and Sylvian fissures). The tumor was sharply defined from the surrounding tissue, and its isolation and extirpation were readily accomplished. The tumor had no relation with the falx and was moderately adherent to the dura, perhaps had already infiltrated it. Oozing from the tumor cavity was trivial and was readily controlled with hot saline irrigations. The wound was closed. As a precautionary measure, 250 c. c. of citrated blood with 250 c.c. of saline solution were transfused at the conclusion of the operation.

Pathologic Report.—On removal the tumor measured 6 by 8 cm. It was irregular in shape, nodular on the surface and definitely encapsulated, except on a portion of the anterior surface. Pathologic diagnosis: tuberculoma (Spiller).

Postoperative Record.—Immediately after the operation there was a transitory hemiplegia and aphasia. Within a comparatively short time the man could move the arm and leg and could talk. Five days after removal of the tumor an examination of the eyegrounds showed a subsidience of the papilledema from plus 4 diopters to plus 2 to 3 diopters, and the signs of hemorrhages seen before the first operation had entirely disappeared. There was for several weeks an irregular febrile course that could not be accounted for and something seemed to retard the patient's convalescence.

A complete search was made for the primary tuberculous lesion. Dr. Pepper reported that there was no evidence of pulmonary tuberculosis. The urine, however, showed an increased quantity of pus, there was tenderness over the region of the right kidney and muscle rigidity in the upper right quadrant of the abdomen. A provisional diagnosis of pyelitis of the right kidney was made and the patient was transferred to the urological service. The patient died while in this service twenty-two months later.

Comment.—A word might be said about the specimen. It was interesting and unusual from several aspects. Tuberculoma is more common in children, is more frequently found in the cerebellar hemispheres, and is not of large dimensions. In all these particulars the tumor did not conform to type. It developed in an adult, in the left cerebral hemisphere and measured over 8 cm. in one of its diameters—a large tumor. Tuberculoma of the brain is secondary to tuberculous lesions elsewhere. The original lesion may be latent or, as in this case, unrecognized before the cranial exploration. If the patient had tuberculosis of the kidney it could not have been very active for a long period, since the patient was reported in excellent health up to within nine months of his operation.

The method of exposure and of extirpation departed from the routine in two particulars. The operation was divided into two sittings and in the interim a ventriculogram was made. When uncertain as to the finding of a tumor or of one's ability to remove it if found, and when the intracranial pressure is extreme, we dislike to reflect the dural flap until at least an attempt is made to relieve the pressure by withdrawal of cerebrospinal fluid. Puncture of the anterior or posterior horn of the ventricle, according to the location of the opening, should be tried. We failed to reach the posterior horn of the right or left ventricle, either directly or by callosal puncture. Either the approach was misdirected or the ventricles were collapsed. We could have proceeded at once to a lumbar puncture but recognized the incidental risks and decided to determine by ventriculography whether one or both ventricles were distended or collapsed and if distended what their geography was.

After one week's interval the ventriculogram was made and the roentgenogram explained our failure to find the ventricles. The ventricles were collapsed, at least there was no air shadow after 100 c.c. of air had been injected. The preparation for a ventriculogram is not unattended with risk under any conditions and particularly when the intracranial pressure is at a high level. This was realized, but one has to measure and weigh relative values, and a complete survey of the cerebrospinal space seemed desirable before the dura was finally opened. The risks were minimized in preparing for the ventriculogram by withdrawing the fluid intermittently. As from 5 to 10 c.c. of fluid were withdrawn, an equal amount of air was injected. Pressure observations were made frequently with a mercury manometer. Using, as we do, a three way cock, merely by the turning of the handle one may (1) take pressure, (2) withdraw fluid or (3) inject air. By repeated observations on the pressure and fractional withdrawal of fluid we reduced the risks to a minimum. There is an appreciable difference in the risk of fluid withdrawn by lumbar puncture in tumors above and below the tentorium. Below the tentorium we regard it as prohibitive.

Ventriculography is in its infancy; as time goes on we will be able to define with greater accuracy the margin of safety and with the confidence of experience will be able to determine its sphere of limitation. In the case under consideration it served a useful purpose. It was of no assistance in the localization of the growth, but it visualized the condition of the ventricles. Had the ventriculogram been made before the operation, we would have been able to finish at one sitting.

Some might question the propriety of a lumbar puncture at the time of the operation in pretentorial growths, but when simultaneously the pressure is relieved above by the reflection of the dural flap, the conditions responsible for foraminal hernia following lumbar puncture do not prevail.

In intracranial procedures mastery of the problems of pressure is essential to the safe conduct of the case. It is in this aspect of the day's work that judgment and experience count.

The condition was far advanced in this case when the man came under the observation of one of us (Spiller). In addition to the general and focal symptoms of tumor, he had a peculiar constant rhythmical jerking confined to the right side of the mouth, and voluntary innervation increased the jerking, as when he separated his lips in showing his teeth; he could not draw up the right corner of the mouth as well as the left. Occasionally, as in the morning after the night's rest, this rhythmical contraction of the muscles of the right side of the mouth was not so conspicuous but became marked immediately when he answered a question. It seemed as though the left facial center were under continual irritation, and voluntary innervation of this center increased its irritability. The condition was that of epilepsia partialis continua, like that described by one of us (Spiller) in association with Dr. Edward Martin.[1] This case has been referred to by Dr. George Wilson.[2]

In this paper with Dr. Martin one of us (Spiller) was able to give very few references to the literature on epilepsia partialis continua and none to any observations published in English or French. Oppenheim had made a brief mention of it in saying that cases occur in which during the interparoxysmal period muscular twitchings of short duration and without loss of consciousness are observed in one or another part of the body. It had been described by Russian writers—Koshewnikow, Muratoff, and Bechterew, and it had been mentioned by Bruns. In the paper by Spiller and Martin emphasis was laid on the fact that the movements differ from those of myoclonic epilepsy in that the twitchings are limited to a small portion of the body and are usually more intense than in myoclonia. Orlowski had reported a case of epilepsia partialis continua and Choroschko had written a monograph on the disorder, but Bruns was the only one not Russian who had observed this rare type. There seemed to be no lesion discovered in any case, so that the case of Spiller and Martin was the first in which a lesion was actually found and the first therefore to establish clearly a cortical origin for the spasmodic movements. In this case jacksonian convulsions occurred, but independently of these typical attacks the man had twitching confined to the right side of the face lasting an indefinite period, an hour or more at a time and occurring very frequently. There were periods in which he had none of these spasmodic movements, but the movements were almost constantly present and resembled a tic movement or fibrillary twitching. They were not excessive in their intensity and implicated the muscles about the right side of the mouth, about the right eye, the right cheek, the right ear and the right side of the neck. After persisting for some weeks in the face they ceased in this part and were observed in the right upper limb. The whole right upper limb was displaced in these clonic movements,

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1. Spiller, William G., and Martin, Edward: Epilepsia Partialis Continua Occurring in Cerebral Syphilis, J. A. M. A. **52**:1921 (June 12) 1909.

2. Wilson, George: The Diagnostic Significance of Jacksonian Epilepsy, J. A. M. A. **76**:842 (March 26) 1921. (Case 1.)

but the displacement was not excessive, and the spasm consisted of slight twitchings lasting indefinitely as did the previous twitchings of the right side of the face. Dr. Martin operated and small gummas were found in the motor cortex.

A case of epilepsia partialis continua was reported by Burr[3] in 1915.

The rhythmical tremor occurring in the present case of tubercle from irritation of the facial center might be confused with the spasmodic jerking resulting from irritation of the facial nerve, as from a tumor of the cerebellopontile angle. In the former condition the contractions are continuous, there is nothing that can be spoken of as an attack; in the latter condition the contractions occur in attacks and simulate the usual epileptiform facial convulsion from irritation of the facial center; indeed convulsive movements have been attributed to irritation in this part, which in reality were caused by irritation of the facial nerve from a tumor. This occurred in a case studied by Dr. Mills with one of us (Spiller) in 1901. Facial spasms were observed in this case under the care of Dr. Mills, and the notes of this case were employed by Weisenburg[4] in a paper on cerebellar tumors published in 1905. The facial spasm was unilateral and caused the mouth to be drawn forcibly to the left and the left eyelids to be drawn together. Both observers thought a tumor was probably located in the facial center of the cerebral cortex of the opposite side and operation was performed; later a cerebellopontile tumor was found. At that time the diagnosis of cerebellopontile tumors was not so well understood as it is now, and facial spasms occurring in attacks had never been reported by any one as a result of irritation of the facial nerve from tumor of the cerebellopontile angle. This patient was completely deaf in the ear on the side in which the facial spasms occurred, but four years before coming under observation the ossicles of the ear on this side had been removed on account of annoying tinnitus, but without the desired result.

In Weisenburg's Case 2 the spasms were limited to the entire distribution of the facial nerve, these coming on without any warning, being first tonic in character and lasting from half a minute to a minute. The lesion was not found. In another case reported by him in which a tumor was found in the right cerebellopontile angle the upper branch of the seventh nerve had been cut, and there had been some return of power in the distribution of this branch. The man had involuntary twitchings in the middle and lower facial distribution; these were sometimes fibrillary, sometimes coarse and spasmodic and were caused by pressure on the seventh nerve. Spasms of the tongue, apparently chiefly right-sided, were observed in this case.

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[3] Burr: Am. J. M. Sc. **149**: No. 2, 1915.

[4] Weisenburg, T. H.: Diagnosis of Tumors and Other Lesions in the Cerebellopontile Angle, J. A. M. A. **50**:1251 (April 18) 1908; Cerebellopontile Tumor Diagnosed for Six Years as Tic Douloureux, ibid. **54**:1600 (May 14) 1910. New York M. J. Feb. 11 and 18, 1905.

In a patient studied by one of us (Spiller) in 1915 convulsive movements occurred in the left side of the face lasting only a few minutes. A tumor was found in the cerebellopontile angle, and a similar case has been observed recently.

Cushing[5] reported before this Association in 1916 two cases of facial spasm caused by peripheral irritation of the facial nerve from a tumor of the cerebellopontile angle. One of these cases is described in detail. It had been previously diagnosed as a case of focal epilepsy from a lesion of the contralateral cerebral cortex. He states that in a series of about fifty tumors in this angle this irritation symptom in a corresponding degree had not been observed. He refers to a paper by Stewart and Holmes in which it was said that evidence of direct irritation of the facial nerve rarely occurs though twitching may occasionally be observed. Cushing describes the spasm in one of his cases as a twitching of one side of the face beginning as a blepharospasm of mild degree coming on a few times a day and lasting for a few seconds. The attacks gradually increased in number, intensity and surface extent until the spasmodic movements became very severe, coming on every ten minutes or so and persisting for a full minute or two. They began with a twitching and drawing of the muscles about the eye, and gradually the whole side of the face was pulled over in a grimace and the surface muscles of the neck (platysma) were affected. The patient stated that in a few severe attacks the left arm and leg mildly participated in the attack and that the left arm and hand felt numb on several occasions, but this Cushing thought was an erroneous statement. The facial spasms were precipitated by talking, laughing or excitement. The man had no control over the spasms and no weakness of the facial nerve supply. The spasms had existed for nine or ten years. A cerebellopontile tumor was found at operation. As the man lived, the cerebral cortex was not examined, but the facial spasms gradually subsided after the operation.

There may be difficulty in diagnosing between the convulsive movements produced by irritation of the facial nerve and those from irritation of the cortical center for the face, as in epilepsia partialis continua. The careful study of the case will usually permit the correct diagnosis, especially the association with other symptoms. In the form from cortical irritation the twitching is likely to be confined to the lower part of the face, as in Pönitz's case to which reference is made further in this paper, whereas in the form from irritation of the facial nerve the whole distribution of the facial nerve is likely to be affected from the beginning. The differential diagnosis may, however, be complicated.

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[5] Cushing, H.: Convulsive Spasm of Face, J. Nerv. & Ment. Dis. **44**:312 (Oct.) 1916.

The facial nerve may be irritated to produce convulsive movements of the face without any paralysis of the facial distribution, as in Cushing's case and one observed by one of us (Spiller), or there may be a statement, possibly erroneous, of convulsive movements in muscles outside of the facial distribution; this leads to confusion. It seems singular that convulsive movements of the face from irritation of the facial nerve occur relatively seldom, and yet the number of cases of tumor of the cerebellopontile angle is considerable.

CASE 3.—*Summary*: *Accurate localization and successful extirpation of an encapsulated glioma from the left cerebral hemisphere; recovery.*

History.—R. McK., aged 30 years, was referred to the neurosurgical service at the University Hospital by Dr. T. P. Farmer of Syracuse, New York, who realized the possibility of a brain tumor, an opinion subsequently confirmed by operation. The patient had had the usual diseases of childhood, including scarlet fever, and appendicitis and catarrhal jaundice; otherwise his health had been good. He had been married two years; his wife and one child, aged 16 months, were well. He denied venereal disease.

Eighteen months prior to admission the patient had a convulsive attack while asleep. It was noted that during the attack he lay on his face moaning "with the left foot out of bed shaking." In two weeks he had another attack, this one during the day. He lost consciousness and foamed at the mouth. Two weeks later he had another attack, and during the first twelve months he had twelve convulsive seizures. During the past six months the seizures had been more frequent, two or three a day, but of quite a different character. In these he did not lose consciousness; the convulsions always began in the fingers of the right hand; the fist was clenched and the arm abducted to a right angle with the body. The muscles of the right side of the neck were involved in the tonic spasm. He had never bitten his tongue, nor had there been any involuntary evacuation. With the exception of these attacks, he felt perfectly well.

After admission to the hospital, the convulsions continued; there was in rapid succession flexion of the fingers, wrist and elbow. The arm was then raised to a right angle with the body, and the muscles of the neck participated in the clonic spasm. Consciousness was preserved.

Physical Examination.—Thorax: There was slight impairment on percussion at the left apex; inspiration was slightly prolonged and harsh. The area of cardiac dulness was normal; there were no cardiac murmus.

Cranial Nerves: There was no impairment of function.

Motor Function: In all movements the right forearm and right arm were distinctly weaker than the left; there was no difference in strength of movement between the right and left lower extremities.

Sensory Function: This was unimpaired.

Reflexes: The biceps and triceps tendon reflexes were exaggerated on the right side; the patella tendon reflexes were equal and normal. There was no ankle clonus and no Babinski sign.

Coordination and equilibrium were undisturbed.

Ophthalmic Report: The pupils were equal and regular in outline and reacted equally to light and accommodation. There were no ocular palsies and no nystagmus. The fields were not contracted and there was no papilledema.

Miscellaneous: The Wassermann, globulin and colloidal gold tests of the spinal fluid were negative. There were 8 cells per centimeter. The Wassermann reaction of the blood was negative, hemoglobin 81 per cent. and the white blood cells numbered 7,900. The roentgenogram was negative.

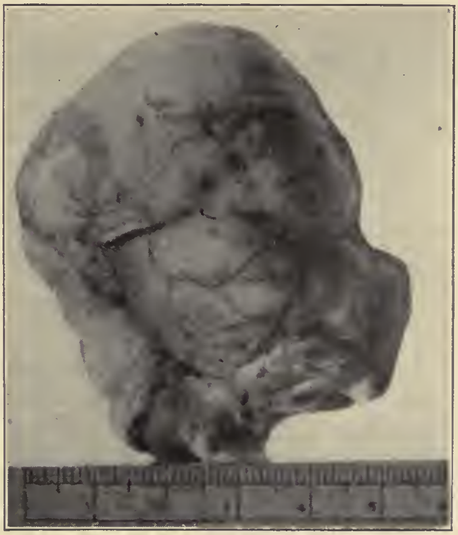

Fig. 3.—Encapsulated glioma.

Summary of Physical Findings: The findings were (1) initial convulsive seizures general in character; (2) later substitution of these with typical jacksonian seizures (right); (3) weakness of the right arm, and (4) exaggerated biceps and triceps tendon reflexes (right).

Preoperative Diagnosis: Tumor motor cortex (left).

Operation.—Dr. Frazier and staff performed an exploratory craniotomy. Ether anesthesia was used and a flap was reflected for exposure of the left motor cortex, two-thirds in front and one-third behind the Rolandic fissure. As soon as the cortex was exposed the lesion was seen in the upper half of the field, in front of the Rolandic fissure. Closer examination revealed a sharp line of demarcation between the tumor and the brain tissue. Around the margins of the tumor as exposed the pial vessels were ligated and with gentle dissection the tumor was freed from the surrounding tissue and removed. The tumor extended 5 cm. below the surface and inward as far as the median line. Its point of origin was undetermined, although at the operation we suspected the falx. Following the removal of the tumor bleeding was trivial; there were but three bleeding points in the tumor bed and these were controlled with muscle grafts. The tumor measured 5 by 6 cm. The wound was closed without drainage.

Pathologic Diagnosis.—Grossly the tumor resembles an endothelioma but histologically it is a glioma, apparently well defined, but not entirely so (Dr. W. G. Spiller).

Postoperative Course.—Immediately after the operation the patient had aphasia and hemiplegia. Within a week, however, he began to recover from both and eventually fully recovered his power of speech and the power of the right leg. The movements of the right arm were still impaired and the limb somewhat spastic. When the patient was examined within a year of the operation, he had had four convulsive seizures; the first two occurred a few months after the operation and were jacksonian in type. There was twitching of the right side of the face and a burning, throbbing sensation in the right arm. In the first convulsion he lost consciousness, in the second he did not.

The second two convulsions occurred nine months after the operation. While talking, speech suddenly failed him, he felt a tense drawing feeling in the right arm and leg, the right arm was abducted and the right leg extended. He remained unconscious for ten minutes. Immediately after he went to bed and within a few moments, a second convulsion, similar to the first, occurred.

Nine months after the operation speech was undisturbed and the power of the leg was restored, but the arm continued weak and spastic. The eyegrounds were normal.

Comment.—The facility with which tumors may be localized when originating in or over the motor cortex is a point in their favor from the standpoint of operability. One might presuppose that in the majority of cases an early diagnosis would be established when the tumor was in its incipiency. Such is not always the case, however. In two of the cases in this series, Case 2 and Case 3, the tumors were of large dimensions and the duration of the symptoms only eighteen months in one and nine months in the other. The origin of these tumors, as to time, is, of course, a matter of conjecture, but as a rule tumors of the brain are slow in growth and one might presuppose with reason a growth 6 cm. or more in diameter might have been several years in process of development.

The history of the convulsions in this case is interesting. For the first twelve of eighteen months prior to the operation they were general in character with loss of consciousness but without focal signs; in the last six months conditions were reversed; consciousness was preserved and the convulsions were typically jacksonian. One can readily account for jacksonian seizures in a lesion pressing on or actually infiltrating the cells of the motor cortex; but in the absence of signs or evidence of increased pressure, without distention of the ventricles, one wonders why the incipient convulsions should have been general and attended with lost consciousness, unless they were caused by general increase of intracranial pressure before the motor cortex of one side was irritated by the tumor.

As in many pretentorial growths, there were no pressure signs; neither headache nor papilledema. This implies of course that the outlet to the ventricles had not been closed and as yet there was no internal hydrocephalus, the most common cause both of headache and papilledema in tumors of the brain without regard to location.

The pathology of this tumor is of unusual interest. In his report Spiller referred to the growth as having the gross appearances of an endothelioma, and so it appeared to the operator. But the histologic examination revealed a glioma, so that we were dealing with a definitely encapsulated glioma, a lesion quite unique in the experience of our clinic. Not only were we deceived in the naked eye appearance of the growth, but as well in our assumption that it had its origin in the falx. With regard to encapsulation of gliomas, of course one often sees what appears to be a distinct wall surrounding a cyst of gliomatous origin. But in this case there was no cyst, and in all the gliomas hitherto exposed in our clinic there have been ill defined margins as the infiltrating tumor invaded the surrounding tissue. We have never seen a definite encapsulation such as in the specimen under consideration. There was, to be sure, a small area on the surface of the growth without a capsule, but we are disposed to believe that in removal this portion of the capsule had become detached.

The postoperative complications, aphasia and hemiplegia, did not disturb us at first. The trauma and edema incidental to the removal of the brain tumors often cause transitory interruption of function. In so far as the aphasia and leg were concerned, the recovery of function in both was no exception to the general rule. The persistent weakness and spasticity of the arm imply, however, irreparable injury.

Whether the convulsions subsequent to the operation were caused by a recurrence or by cerebral trauma is still an open question; either of course would explain them; but one would hardly expect a recurrent growth within five months of the operation, the date of the first convulsion, to be of sufficient size to be responsible for them. We can, with just as much reason, attribute the convulsion to the immediate effects of the operation trauma, hemorrhage and cicatrization. In all patients with epileptic seizures we give luminal, 1½ grain, in the evening, from the day of the operation for a period of six months to a year or longer.

CASE 4.—*Summary: A calcified endothelioma intrahemispheric, exposed at operation and treated by radium implantation with relief from convulsions and headaches.*[6]

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[6] While this tumor was not removed, this case is included in the series because of the relief which followed the exposure of the tumor and direct radium implantation.

History.—O. T., aged 37 years, was admitted to the neurosurgical service of the University Hospital, April 11, 1920, referred by Dr. M. H. Tallman. She had had measles and chickenpox in childhood and diphtheria at the age of 20. She had had two miscarriages, one before and one after the birth of a living child (now 3½ years old). A maternal aunt had epilepsy. Otherwise her family history was negative. She was married five years ago. She was engaged in housework and her living conditions were wholesome.

Ten years before admission the patient first complained of momentary attacks of dizziness, occurring about seven times a day. Three months after the onset of these she noticed that during an attack the left knee became weak and she nearly fell. These attacks continued for four months, at the end of which time she had her first severe convulsion. The convulsions were preceded by a choking sensation, the left arm was abducted, the lower left limb drawn up, and she lost consciousness for about ten minutes. She came out of the convulsions groaning, but did not fall asleep. Only the left side of the body was involved in the convulsions. At first they occurred about once a month, then gradually they became less frequent and after her first child was born there was an interim of a year and nine months in which she had no attacks at all. About two months before admission the attacks recurred, lasting a few minutes; in these attacks her left upper and lower limb felt numb, and she could not raise her left arm or foot. During this period for the first time she complained of blinding headaches, occurring about once a week, sometimes in the right temporal region and sometimes in both.

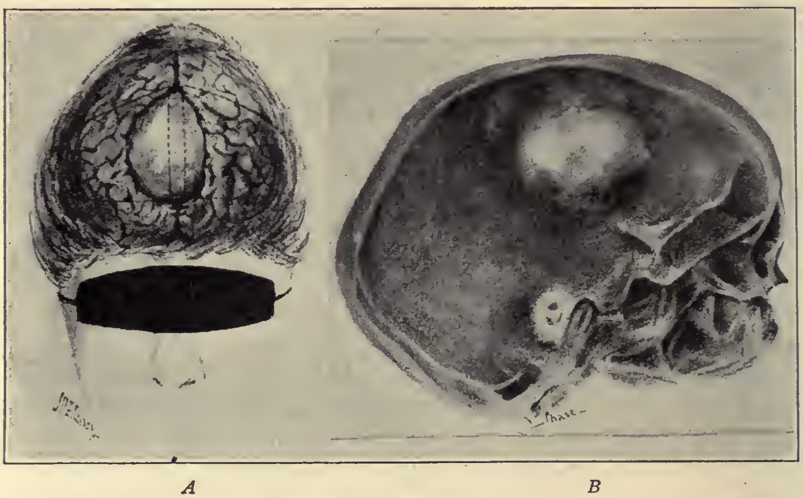

Fig. 4.—*A,* schematic drawing showing relation of tumor to longitudinal sinus; *B,* drawing of roentgenogram showing calcification of tumor.

Physical Examination.—Examination of the scalp, nares, teeth and tonsils was negative. The post-cervical glands were palpable. The lungs were normal. There was a systolic murmur over the aortic area of the heart. Blood pressure was: systolic 100; diastolic, 70. Examination of the abdomen was negative. Urinalysis: The specific gravity was 1.024. The urine was acid and there was a trace of albumin. There was no impairment of motion in either arm or leg, and normal sensation was preserved. Both the right and left patellar reflexes were exaggerated.

The Wassermann test of the blood and spinal fluid was negative.

Eye Examination.—Muscular movements were free and equal. The pupils were equal and regular and reacted to light and accommodation. Right eye: The media were clear and the disk margins were veiled with fine striations running out into the retina. The veins were overful and slightly tortuous. There were no discrete lesions of the choroid or retina. Left eye: The media were clear and the disk margins obscured. The disk was elevated from 1 to 2 diopters. The veins were full and tortuous. There were no discrete lesions of the choroid or retina.

Roentgen-Ray Examination.—This showed a calcified tumor, extending from the vertex more than half way to the base of the skull. It was viewed anteroposteriorly, in the midline position, extending more to the right than to the left. There was no evidence of intracranial tension and no sellar deformation.

Preliminary Diagnosis: Calicified endothelioma.

Exploratory Craniotomy.—First Stage: Flaps were reflected on both right and left sides, uncovering the region of the tumor and the longitudinal sinus. The patient's condition was not tolerant. No attempt was made to remove the tumor at this sitting.

Second Stage: Nine days later the flaps were again reflected and the tumor exposed. The situation of the tumor with relation to the longitudinal sinus made it evident that a section of the sinus would have to be removed with the tumor. Furthermore, the tumor had infiltrated the overlying dura so that to extirpate the tumor successfully would require removal en bloc of the tumor, overlying dura and a portion of the longitudinal sinus. A dural incision circumventing the tumor was made. The longitudinal sinus, anterior to the tumor, was divided and closed with silk ligatures. Though there had been little bleeding and a minimum of trauma, the patient's condition gave us concern. The pulse rate was 150 and of poor quality. No further steps at extirpation were taken for the moment. Exploring with the finger the tumor was found to be attached to the falx and to extend almost (if not to) the base of the skull. So firm were its attachments that there was no play either in the lateral or anteroposterior directions. On account of the patient's condition and because the tumor was so firmly attached, further attempts at removal seemed too hazardous. The dural and scalp incisions were closed. Direct transfusion was made of 500 c.c. of citrated blood.

Radium Implantation.—May 14, 1920, eighteen days after operation, under procain anesthesia, a small area of skull overlying the tumor was removed with rongeur forceps. A container with radium, 50 mg., was implanted in the tumor. In attempting to direct the container toward the center of the tumor, it was obstructed by some impermeable substance (calcified areas in the tumor). The container had to be directed forward to imbed it completely in the tumor. The radium was removed in eighteen hours. The dosage was 900 mg. hours.

Postoperative Record.—Immediately after this exploratory craniotomy the patient developed a hemiplegia from which she had largely recovered at the time of her discharge. Thirteen months after the operation, her physician wrote that the patient was feeling very well, that while she had attacks of dizziness once in a while, she had had none of the convulsive seizures since her discharge. In response to a suggestion that she return for a second radium treatment, the patient stated that in view of her improvement, she did not feel justified in incurring the expense of the journey across the continent from her home in the West to the hospital.

Comment.—There are many points of interest in this case. For almost ten years the history of the growth was characterized solely by unilateral convulsive seizures involving the left arm and the left leg. The fact that after the seizures the patient experienced weakness in the arm and leg had aroused a suspicion of tumor. It was not until well within the tenth year of her illness that other focal signs developed, such as transitory periods of numbness in the left arm and left leg and, for the first time, blinding headaches. In spite of the duration of the lesion and of its huge proportions, in spite of the blinding headaches, there was little evidence of increased intracranial pressure in the condition of the optic disks. It is noted in the eye examination that there was an elevation of only from 1 to 2 diopters in either disk.

Had there been any doubt as to the diagnosis, and there could have been little, the evidence of the roentgenogram was conclusive. From the shadow of the tumor in the roentgenogram one is justified in making a preliminary diagnosis of calcified endothelioma, although in another case, a tumor quite as vividly revealed in the roentgenogram proved to be an enchondroma.

The location of the tumor, practically intrahemispheric and beneath the longitudinal sinus, necessitated a two flap operation, one reflected to the left and one to the right. This two flap operation I found gave a splendid approach and exposure. The immobility of the tumor is difficult to account for, unless it was adherent to the base of the skull, and yet we know that many of these midline endotheliomas take their origin from the falx. Except for this unusual immobility there appeared no obstacles to its removal, and in a more robust patient an attempt would have been made to mobilize it. After the operation it occurred to me that we might have attempted a subcapsular extirpation such as we practice at times in cerebellopontile angle tumors.

In this particular case, however, I doubt whether the patient would have survived any radical undertaking. The intolerance to surgical intervention of patients with tumors of long standing is an interesting speculative study. From the very beginning of an operation, the pulse, as in this case, becomes accelerated; a rate of 140 to 150 is common. The mere reflection of the osteoplastic flaps seemed a sufficient undertaking for one sitting and a two stage operation seemed imperative. At the second sitting, even before any attempt at demobilization of the tumor had been made, there was reason for anxiety. The disturbance of the cardiac mechanism in brain tumor cases I have attributed, rightly or wrongly, to a delicately balanced blood supply of the medullary centers. How else can one account for the rapidity of pulse rate and the falling of blood pressure? In the absence of serious hemorrhage there apparently is no other way of accounting for them.

Case 5.—*Summary: Successful extirpation of an endothelioma 3 by 2 cm. from a patient whose illness began with fleeting attacks of aphasia, followed by jacksonian seizures; recovery.*

History.—C. M., aged 17 years, was referred to the neurosurgical service at the University Hospital by Drs. William G. Spiller and H. M. Fussell. When 16 years of age, she first consulted one of us (Spiller) Sept. 21, 1918. About a year previously she suddenly lost the power of speech for about three minutes. while walking along the road. She was frightened. At first she stuttered and then was entirely unable to speak. There seems to have been no other symptom at this time. About a month later she again lost her speech suddenly, for about a minute and a half, without any premonition or stuttering, and again she had no other symptom. She tried to write while she was unable to speak this second time but could not form any letters during this period. She had lost her speech about six times in all for a brief period. About the middle of July, 1918, while in a field, objects suddenly seemed to be going around; she fell to the ground and was unconscious about ten minutes. About a month later, while riding horseback, she suddenly lost her speech; she got off the horse and laid down on the ground and may have lost consciousness. In the later attacks with loss of speech she had a peculiar sensation in her right forearm and hand, and she could not move the right hand at these times, but the hand twitched and the two times she lost consciousness the right side of the mouth twitched.

About three weeks before coming for examination her mother found her making a noise with her mouth, she was somewhat rigid, the right side of the mouth twitched and she was unconscious. In this attack there was a numb feeling in the whole right side, face, limbs and trunk. Dr. Zentmayer in September, 1918, found the veins of the left retina a little fuller than normal.

Attacks occurred which were observed by her parents. They began, the patient said, with a loss of voice, when she would lie down; a twitching of the right arm and the muscles around the mouth was noticed, which were drawn to the right side. In the early attacks she thinks the right leg moved but this is questionable. The attacks occurred about a week apart; the duration was the same, from one to three minutes, and they were characterized by twitching of the right arm and mouth muscles. Each attack began with what she felt was a loss of voice, which forewarned her so that she lay down. In the present month she had had two attacks and the beginning of a third, which apparently was abortive. In these attacks she had held the right hand firmly on her breast and the arm had not appeared to move, but the muscles of the mouth trembled and were drawn to the right. These last two or three attacks had not lasted for more than a minute or two and with less convulsive movements, the arm apparently not taking part.

Repeated examinations of the eyegrounds showed no positive change. Her muscular tone seemed as good as ever, there was not the slightest diminution of power in the right arm, and the grip of the right hand was perhaps unusually strong. Her general health and spirits, if anything, had been better this month than in the past two. She had as lively an interest in affairs as usual, was planning to return to school, and had been allowed to go to entertainments and take part in occasional dances as usual.

Preoperative diagnosis: Tumor of the motor cortex, left, near or in Broca's area.

Operation.—A craniotomy was performed by Dr. Frazier and his staff. Ether anesthesia was used. An osteoplastic flap was reflected, designed to expose the motor zone and the region of Broca. When the dural flap was reflected there was seen just above Broca's region an area of minute dimensions, less than a centimeter in diameter, that differed in appearance from the normal brain tissue. The nature of it at first was not realized, but on closer examination it proved to be a tumor which without difficulty was separated from its bed and removed. The tumor was quite the smallest of any in the experience of our clinic, measuring in its largest dimension not more than 3 cm. The operation was free from any technical difficulties. The wound was closed without drainage.

Pathologic Diagnosis: Endothelioma.

Comment.—The attacks of loss of speech indicated a lesion of the left cerebral speech area, and they seem to have been purely of the motor type, as no sensory aphasia was noted. At first there was no other disturbance than that of motor speech and writing, and as the tumor was very small and could not have caused much pressure the loss of speech was a sharply defined focal symptom. It would seem to indicate a lesion of Broca's area. The loss of speech was complete temporarily, and this is the form of aphasia to be expected in a lesion of Broca's area. It is very different from that occurring with a small lesion of the sensory speech area. In this improper words are used, the patient cannot think of the words he wants to use and uses others incorrectly. This has been seen several times by one of us (Spiller) in tumor of the left temporal lobe. It is the earliest manifestation of beginning word deafness, and the improper use of words precedes distinct signs of word deafness. Indeed this improper use of words in the early stage of lesions of the left temporal lobe may be so slight that a search must be made for it. A patient now under observation from whom a large tumor of the left parietal lobe has been recently removed by Frazier understood everything said to him and spoke very well, but he at times could not think of the words he wanted to use, especially the names of objects, and showed beginning word blindness in that he confused figures and did not understand everything he read.

The loss of speech in this young girl was not caused by a temporary paralysis of the face center, as it does not appear likely that a temporary paralysis of all the muscles of speech could result from a lesion of the face center of one side. This probably would result only from a bilateral lesion of the center. The agraphia also is evidence of a true motor aphasia.

Transitory motor aphasia with convulsive movements of the right side of the face and right upper limb later constituted the whole clinical picture. The father of the patient was a physician and understood the focal importance of these symptoms. Rather than permit his daughter to continue having these distressing attacks he consented to an operation, with the discovery of a minute well defined endothelioma in or very close to Broca's area, distinctly separated from the underlying brain, and its successful and complete removal. Soon after the operation all convulsions ceased and no more have occurred. The recovery is complete in every detail and there seems to be no probability of recurrence. Not a single symptom persists.

The occurrence of complete transitory motor aphasia in a young person may therefore be a focal symptom of importance. In an elderly person it may result from arteriosclerosis, paresis or some other cause.

A case very similar to this one, except in regard to the aphasia, is reported by Pönitz.[7] Aside from some hysterical manifestation, the important symptom was clonic convulsion of the left side of the face, except the upper part of the face, occurring in attacks, of two years' duration. Later the left upper limb became involved in the convulsions. A solitary tubercle the size of a cherry stone was found in the right facial center.

Case 6.—*Summary*: *The successful removal of a tumor, with the characteristics of a fibroma, from the right cerebellar fossa. The patient recovered and continues free from symptoms three years after the operation.*

History.—Mrs. O., aged 45 years, was referred to the neurosurgical service of the University Hospital by Dr. G. A. Young of Omaha, to whom I am indebted for the following history:

Four years ago the patient began to complain of dizziness and unsteadiness of gait; the latter was slight at first but gradually became more pronounced, and it was constant. The dizziness was not associated with rotation of object or self. Eighteen months ago she began to complain of headache and vomiting periodically. Some months later she experienced a sense of numbness in the right side of her face.

At the present time there is a definite hypesthesia with loss of the conjunctival reflex. There is a slight weakness of the right facial distribution but no disturbance in either of the cochlear or vestibular branches of the auditory nerve. The visual fields are slightly contracted and there is an elevation of two diopters in each disk. There is no disturbance of the motion or sensation in any of the four extremities, and the reflexes are normal. There is a suggestion of adiadokokinesis in each upper extremity. She speaks of her left side as being her good side, without being able to say precisely in what way it is the better. At the present time she has comparatively no headache, but nausea and occasional vomiting persist.

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[7] Pönitz: Deutsch. Ztschr. f. Nervenh. **67**: Nos. 1 and 2, p. 89.

At the present time the Bárány tests show an irregular spontaneous past pointing in both arms which may be either to the right or left; both vertical and horizontal canals react to the caloric test in both right and left ears. In the turning test she shows a normal nystagmus reaction in either direction, the past pointing is somewhat indefinite, but she past points in the right direction when turned to both left and right. The falling reaction is normal also to the right and left. The subjective sense of after-turning vertigo has a duration of about twenty seconds when turned to both right and left.

Preoperative Diagnosis: Tumor of the right cerebellar chamber.

Operation.—A suboccipital craniotomy was performed by Dr. Frazier and his staff. An encapsulated tumor was found and removed from the right cerebellar fossa.

Pathologic Diagnosis: Fibroma.

Unfortunately the record of the physical examination and the operative details have been lost, so that we are able to present only the foregoing facts. The patient recovered and is entirely well, three years after the operation.

CASE 7.—*Summary: Seven years after the onset of symptoms and one year after a suboccipital decompression, extirpation of glioma from left cerebellar fossa; three courses of radium therapy before operation, during which tumor became encapsulated. Three years after extirpation of growth no evidence of recurrence.*

History.—E. E. S., aged 38 years, was referred to me by Dr. Charles F. Neu of Indianapolis. The family, social and previous history were without interest.

Six years ago the patient's wife observed that his gait was shuffling, but no other symptom was noticed until three years later when the hearing of his right ear became impaired. Two and a half years ago he noticed numbness in his lips at the angle of the mouth, and an abrasion on the tongue which he had bitten unknowingly. About the same time the hearing of the left ear became impaired and sensation in the left side of his face was lost. In walking he staggered to left and occasionally lost his balance and fell. His eyesight was not as good as it had been and headaches became more or less constant. During the preceding eighteen months he had attacks of vomiting and, because of his disability, had to give up his position as a school teacher. Within the year and before he came under our observation, a suboccipital decompression was performed. This relieved his headaches in part, but the patient thought his incoordination was more marked. Recently he had had difficulty in talking and swallowing, occasionally choking when swallowing liquids.

Physical Examination.—Head: There was a moderate hernia at the site of the previous decompression.

Cranial Nerves: Second, Third, Fourth and Fifth Nerves: The pupils reacted to light and accommodation. Ocular rotations were full. The vision in the right eye was 6/12; the papilledema plus 5 diopters. Vision in the left eye was 6/15; papilledema plus 6 diopters. The disk was rather spread out and more succulent than one would expect in a choking of long standing. It may imply an acute exacerbation (Holloway). The fields showed some irregular contraction but nothing of localizing significance. There was horizontal nystagmus on lateral rotations; on upward and downward rotation the movements became vertical. Fifth Nerve: There was a sense of numbness in the left trigeminal distribution. Seventh Nerve: The upper branches were slightly involved. Eighth Nerve: There was marked impairment of hearing in the left ear. There was vertigo on stimulation, with complete absence of past pointing of the left arm to the left. Impaired response from the right or functioning ear was noted. The right labyrinth was not affected. The findings suggested a lesion of the left cerebellopontile angle with extension to the left cerebellar hemisphere and brain stem. Ninth Nerve: There was difficulty in swallowing. Twelfth Nerve: There was no deviation of the tongue when protruded.

Extremities: The grip of both hands was firm and equal. No weakness was present in the lower extremities.

Reflexes: Biceps, triceps and patellar tendon reflexes were more marked on the left than on the right side.

Coordination: Movements of the left arm and leg showed marked incoordination. Adiadokokinesis was marked.

Miscellaneous: The blood pressure was: systolic 135; diastolic 70. The red blood cells numbered 2,510,000; the white blood cells, 8,240; hemoglobin 92 per cent. The urinalysis was negative. Roentgenogram: The posterior clinoid processes were exceedingly thin. The sella turcica was not enlarged. The right and left internal auditory meatus were of equal dimensions.

Preoperative Diagnosis: Tumor of the left cerebellopontile angle.

Operations.—A suboccipital craniotomy was performed by Dr. Frazier and his staff. Ether anesthesia was used, endopharyngeal. On reflection of the flaps a tumor was exposed occupying the entire left fossa. It had the gross appearance of a glioma and contained a large cyst. Extirpation was considered impossible and 50 mg. of radium were inserted into the cavity of a cyst to be removed in eighteen hours. The wound was closed.

Before the patient was discharged, radium was applied by Dr. Pancoast over the surface in the suboccipital region. Three months after the operation the patient returned for observation and a second course of radium therapy. His sight, hearing and articulation were improved. Incoordination was about the same. There was a marked subsidience of the choked disk and the nerve margins were readily discernible. Elevation of the right disk was plus 2 diopters; of the left plus 3 diopters.

Eight months after operation a third course of radium therapy was given. He was totally deaf in the left ear but hearing in the right ear was much improved. There was no particular change in the condition noted except in the movement of the left leg and vision. The heel to knee test (left) revealed no ataxia. Vision in the right eye was 6/9; in the left 6/9 (previous to operation it was 6/20-6/15). The fundi level on either side was plus 1 diopter. The disks were somewhat obscured on the nasal side but were visible in the temporal margins.

Ten months after operation, the patient returned with evident aggravation of symptoms, particularly noticeable in the three previous weeks. Headache had returned, hearing in the right ear again becoming impaired; lately he had been swaying to the right in walking, previously only to the left. His vision was not as good; it was 4/15 in each eye, the disks were plus 4.5 diopters. In other respects there had been no appreciable alteration in the clinical picture. Because of the evident recurrence and at the patient's earnest request a second operation was agreed on.

Operation 2.—A suboccipital exploration was made. When the musculo-aponeurotic flap was reflected, the dura not having been replaced at the previous occipital decompression, the tumor presented itself. But in appearance it differed in that now it was well encapsulated. Complete extracapsular extirpation did not seem feasible; the capsule was split and the contents removed. Grossly the tumor had the appearance of a cystic glioma. Again 50 mg. of radium were introduced within the capsule, to be removed in eighteen hours. Wound closure was made.

Pathologic Diagnosis: Glioma.

Final Note.—Three years after tumor extirpation and nine years after the appearance of the first symptom, the patient returned for examination. He reported that he had been active in business, and had less difficulty in walking; the hearing in the right ear was much more acute, and he had had no nausea, vomiting or headache. On examination there was ataxia and dysmetria in the left upper and lower extremity; the nystagmus, particularly in extreme positions, persisted. Vision in the right eye was 6/9 plus 2, in the left 6/12 plus 2. The margins of the disks were clear; no papilledema.

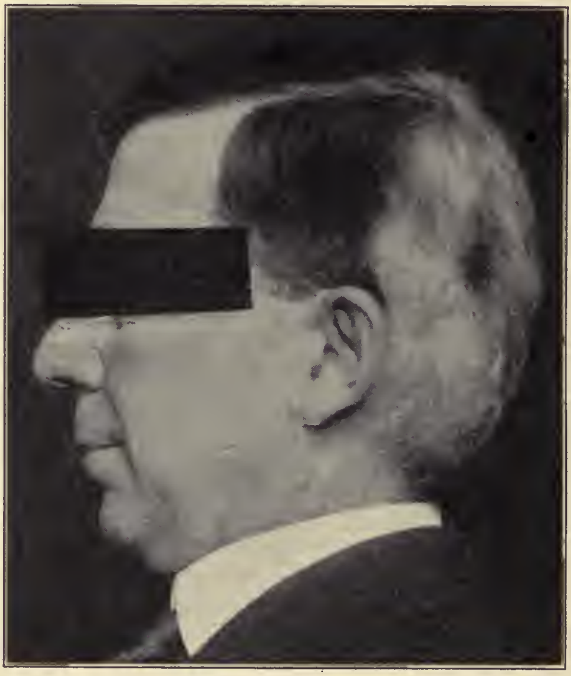

Fig. 5.—Photograph of patient taken nine years after operation. In the area to which radium was applied the hair is white.

Résumé of Case.—Nine years ago, the first symptom, disturbance of the gait, appeared. Six years ago there was disturbance of hearing followed by numbness of the face, failure of vision, vomiting, headache, difficulty in swallowing and speech, dysmetria, etc. Four years ago at a suboccipital decompression the tumor was exposed but not removed; radium therapy. Three and one half years ago radium therapy (course 2) was utilized. Three and one quarter years ago a third course of radium therapy was given. Three years ago extirpation of the growth was performed and a fourth course of radium therapy was given. At present there is no evidence of recurrence.

Comment.—The larger one's experience with tumors of the brain the more one is struck with their rate of growth. How slow it is compared to malignant tumors elsewhere. Even is this so, as in the case under discussion, with gliomas which in their cellular characteristics resemble the sarcoma group. In this case there were signs of a tumor developing six years before the operation, and though radical extirpation was not possible in the surgical sense, there has been no evidence of recurrence three years after removal.

The localization of the growth was not difficult. In the evolution of the clinical picture the first symptom was disturbance of gait and some time elapsed before there was any other evidence of tumor. The three year interval between the initial symptom and impairment of hearing was more than suggestive of a lesion originating in the cerebellar hemisphere rather than the cerebellopontile angle. At least this interval excluded the likelihood of an acoustic tumor. As a matter of fact, there was never complete nerve deafness. One of the earlier symptoms was a sense of numbness in the face, on the affected side, and yet at no examination could one demonstrate any loss of appreciation of tactile or pain sense.

What may have been the effect of radium therapy on the tumor growth is a matter of speculation. At the second operation there was a definite encapsulation of the growth, not observed when the tumor was originally exposed at the decompressive operation a year before. That radium incites hyperplasia of connective tissue elements is generally acknowledged, but this would hardly explain the formation of a capsule about the tumor, and connective tissue elements within a glioma are negligible. From what we know of the effects of radium on tumors, we have come to the conclusion that it is more effective in the endotheliomas than in the gliomas.

When an entire cerebellar hemisphere has been destroyed by the tumor one cannot of course expect recovery of function after the tumor is removed. There must persist, as in this case, disturbance of the function, attributable to the hemisphere destroyed. And so while the headache, vomiting, papilledema and to some extent the deafness disappeared (all pressure phenomena), the ataxia, dysmetria, disturbance of equilibrium and gait persisted.

As to the operability of gliomas, in many instances they have been regarded by us as inoperable, particularly diffuse subcortical infiltrating growths of the cerebral hemisphere. But in this case the tumor occupied the entire left cerebellar fossa; one could not see a vestige of the cerebellar hemisphere, and as it appeared well encapsulated the contents of the capsule at least could be extirpated. Even should there be a recurrence at some later date, a second operation should be advised with a reasonable hope of removing the recurrent growth.

Prolongation of life, even after partial extirpation, should be given a prominent place when discussing the pros and cons of surgical interference in brain tumors. To be able to prolong the patient's life, in a reasonable state of health and activity and at the same time to conserve vision, is in itself a sufficient endorsement of surgical intervention. In this case the patient has had over three years of complete comfort and business activities.

CASE 8.—*Summary: A man, 29 years of age, with a fibroma of the acoustic nerve, accurately localized before operation, subsequently exposed and removed; recovery.*

History.—L. A. S., aged 29 years, was referred to the neurosurgical service by Dr. William G. Spiller with the following history:

Three years ago the patient noticed that he did not hear well with his right ear; and ten months ago his hearing, he observed, had become much worse. Seventeen months ago, when he became tired and nervous he began to have a "roaring" in the right ear. He noticed a peculiar sensation in his gums and cheek on the right side, twenty months ago and fourteen months ago difficulty in writing and shaving. Nothing of consequence developed for almost a year when (five months ago) he began to have trouble in walking, with a tendency to stagger, usually to the right, when walking in the dark or on a narrow path. A month later he first complained of disturbance of vision; he could not focus accurately, particularly when moving about. Objects looked blurred, but there was no dizziness. Now, when reaching for an object his hand miscalculates the distance by an inch or so and in going upstairs he has to concentrate on the movements of his right foot.

The chronologic sequence of events is: Three years ago, there was disturbance of hearing (right); twenty-two months ago, deafness more pronounced (right); twenty months ago, sensory disturbances of gum and cheek (right); seventeen months ago, tinnitus in right ear; fourteen months ago, awkwardness in movements of right hand; four months ago, staggering in walking; three months ago, visual disturbances; at present, awkwardness in movements of the right hand and right foot.

Physical Examination.—Cranial Nerves: The first, third, fourth, sixth, tenth and eleventh nerves were normal. There were no ocular palsies. Nystagmus was present when looking to the right or upward (ophthalmic report). Fifth Nerve: The sensation in his face differed on the two sides. When he was pricked with a pin in the distribution of the fifth nerve he felt it as a sharp prick on the left side, but on the right side he said it felt "muffled," though he knew it was sharp, not dull. The sensation was the same on the two sides in the cervical distribution in the lower part of the face. When he chewed he used the left side because he said the right cheek and gums felt as if they had been burned. The right eyeball and cornea when touched with the head of a pin showed no contraction of the lids; the left conjunctival reflex was normal. When he bit, there was no difference in the masseter or temporal contractions on the two sides.

Seventh Nerve: When he wrinkled his forehead the lines were not quite as deep at the extreme right as on the left side, and there was a gradual lowering of the eyebrow on that side so that in order to keep his forehead wrinkled he had to make repeated efforts, but this was slight. When he showed his teeth both corners of his mouth were drawn back equally, but soon the right side began to relax, and there was a twitching of the muscles in an effort to keep the corner drawn up. The resistance to raising the upper lid when the eye was closed was slightly less in the right eye (Dr. B. A. Randall).

Eighth Nerve: The right ear seemed deaf for all lower tones and even the higher ones were less well heard than on the left. The left ear responded normally to all tests. Past pointing was not spontaneously present, but easily though briefly elicited by rotation or touch. There would seem to be some brainstem lesion anterior to the cerebellar peduncles. The absence of ocular evidence suggested a very small lesion. He could not hear a watch tick close to or even on his right ear. The hearing in his left ear was apparently normal.

Ninth Nerve: The soft palate was normal.

Twelfth Nerve: There was no deviation in the protruded tongue.

Sensation: In the upper extremities, sensation was normal for touch and pain. There was no asterognosis and no loss of sense of position. In the lower extremities there was no loss of sense of position and no disturbance of sensation.

Reflexes: In the upper extremities the reflexes were equal and normal. In the lower extremities the Achilles tendon reflexes were prompt and equal. The right patellar reflex was a trifle prompter than the left. There was no Babinski sign.

Cerebellar Disturbances: There was slight but distinct dysmetria and adiadokokinesis in the right hand. When he wrote he did not slide his right hand along the paper (dyssynergia).

Gait: With his eyes open the gait and station were normal; with eyes closed, the station was good but in walking there was a tendency toward deviation to one side or the other.

Roentgenogram: There was no evidence of any sella deformation. In all respects negative.

The Wassermann reaction of the blood was a doubtful negative. The cerebrospinal fluid was negative.

Résumé of Symptoms: The symptoms were: deafness in the right ear, tinnitus in the right ear, hypesthesia in the right trigeminal zone, facial paralysis partial (right), retinal veins a little larger than normal, dysmetria of the right upper extremity, dyssynergia of the right upper extremity, adiadokokinesis of the right upper extremity, patella reflex a trifle prompter on the right side and deviation to the right in walking.

Preoperative Diagnosis: Acoustic tumor.

Operation.—Ether anesthesia was used, endopharyngeal method. A suboccipital craniectomy was performed with extirpation of the tumor. The bone was removed from over both cerebellar hemispheres, the occipital sinus ligated and the dural flap reflected. There was an increased intracranial pressure, but when the fluid in the basal cistern escaped, the pressure was restored to within normal limits. Displacement of the right cerebellar hemisphere disclosed an acoustic tumor about the size of a grape. The tumor was bisected and removed in segments. Hemorrhage from two points was controlled with muscle grafts. Closure was made without drainage.

At the conclusion of the operation 600 c.c. of citrated blood was transfused.

Postoperative Course.—The patient's convalescence was somewhat prolonged. He was extremely apprehensive throughout. For a long while he complained of difficulty in swallowing and would not take solid food, although he never choked when taking liquid food as one observes when there is any disturbance of the muscles of deglutition. He complained also for some time of mucus collecting in the throat. But apart from these symptoms there were no unusual complications.

Pathologic Report.—The tumor consisted of spindle-shaped cells having a parallel arrangement and forming bundles. The tumor contained a fair amount of connective tissue and some round nuclei, which were perhaps in part at least transverse sections of spindle-shaped nuclei. The tumor was a typical fibroma (Dr. W. G. Spiller), the characteristic tumor of the acoustic nerve.

Comment.—The sequence of events in the early history were characteristic of an acoustic tumor. Of significance in the diagnosis was the initial deafness. Three years before the operation the patient noticed that he was becoming deaf and for many months this was the only symptom. Then followed in sequence the involvement of the sensory root (hypesthesia), tinnitus and the involvement of the facial nerve. After that there were evidences of disturbance of cerebellar function, dysmetria, dyssynergia, adiadokokinesis and staggering. The primal disturbance of hearing, antedating by so many months any other symptom, either of the cranial nerves or of the cerebellum, was of great significance in the differentiation of angle tumors, other than those originating in the acoustic nerve or of tumors of the cerebellum with secondary invasion of the cranial nerves.

Up to the time of operation there had been no obstruction to the outlet of the ventricles, hence the absence of pressure phenomena. Apart from a slight fulness of the retinal veins there were none. The absence of ventricular distention was observed at the operation, since once the fluid in the posterior fossa escaped there was no evidence of pressure, and it was not necessary, therefore, as in many explorations, to evacuate the ventricles. The right hemisphere was displaced without any difficulty sufficiently for the manipulations necessary for the removal of the tumor.

The tumor, as reported, was a fibroma and not as in many instances an endothelioma. But even though it was a fibroma, there is a possibility of recurrence. These two tumors, fibroma and endothelioma, have similar characteristics; each is relatively benign, but each may recur. An endothelioma may recur as a diffuse process and a fibroma may become readily a spindle cell sarcoma (Spiller).

The surgical problem was devoid of any technical difficulties. The accuracy of localization and the character of the tumor rendered the exposure of the growth and its removal a relatively simple procedure.

The resort to blood transfusion after prolonged intracranial procedures, particularly after suboccipital operations, we believe has merit.

It has not been our practice to reserve transfusions for those cases in which hemorrhage has been profuse. This in itself constitutes a positive indication. But when there has been a prolonged anesthesia and a prolonged operative siege we find that the reaction from these after transfusion is prompter, and the convalescence shortened. From the condition of the patient the day after the operation, when transfusion has been practiced, one would not suspect that he had been subjected to a protracted ordeal on the operating table the day before. The value of blood transfusion in this particular field is enthusiastically recognized by all who come in contact with the patient.

ABSTRACT OF DISCUSSION

Dr. Charles K. Mills of Philadelphia said that, with regard to very small growths, he had had several experiences, one of the growths being in Broca's area and another in the parietal region. The paper was instructive in that it supported the older so-called classic view of aphasia as against the hypotheses of Marie. With regard to epilepsia continua, Dr. Mills said that he had had several cases, for one of which he had had a cranial-cerebral operation over the motor area performed by Dr. Frazier. No lesion was revealed. Dr. Mills said he recalled that Dr. Burr had, some years ago, reported a case of epilepsia continua and discussed the subject in a paper. Of course the whole subject belongs to the old and interesting field of accurate cerebral localization.

Dr. Mills remembered quite clearly the case to which Dr. Spiller alluded, in which they had made the diagnosis of a lesion in the facial cortical center, necropsy showing that the lesion was in the cerebellopontile angle. Recurring facial spasm of moderate intensity was a striking feature in this case. The spreading of jacksonian limb symptoms in these cases of cerebellar pontile lesions was, Dr. Mills thought, not difficult to explain, owing to the proximity of the pyramidal tracts.

Dr. H. H. Hoppe of Cincinnati said, in connection with the small tumors of the psychomotor area, that it might be interesting to know that at times they are tubercles, which are arrested in their growth, become calcified and may remain latent for many years.

A patient was brought into the Cincinnati General Hospital with a supposed stroke of apoplexy on the right side, sudden in onset. He died without regaining consciousness. Necropsy revealed a calcified mass, about the size and shape of a peanut, just below the cortex of the rolandic area in the ascending frontal convolution on the left side. The history of the case, obtained from a relative, disclosed that forty years before, the patient, when 16 years of age, had developed gradually, in the course of several months, a right-sided weakness of the body. He recovered from this and for forty years afterward did hard manual labor.

The pathologic examination revealed that the mass was a calcified tubercle surrounded by a cartilaginous envelope.

Dr. Hugh T. Patrick of Chicago mentioned a case of epilepsia partialis continua, in a boy of about 10 years. The first symptom had been intermittent shaking of the right foot. In other words, it continued uninterruptedly for a while and then stopped for a time. The movement very much resembled that of some of the shakers with war neuroses. Another feature of it was that it continued during sleep. When Dr. Patrick saw the boy he was sound asleep but the right leg was in this continuous clonic spasm. By that time the boy had also developed typical jacksonian fits, beginning in the right leg. The shaking was interrupted by a more tonic spasm, succeeded by a clonic spasm that spread over the entire right side. It was a case of cortical tumor of the left hemisphere.

Dr. William G. Spiller of Philadelphia said he was aware of the paper written by Dr. Burr, which was published some years after the one by Dr. Martin and himself, and a reference was made in the paper by Dr. Frazier and himself to Dr. Burr's paper.

ACUTE ASCENDING MYELITIS OF THE INFECTIOUS TYPE

ITS POSSIBLE RELATIONSHIP TO POLIOMYELITIS IN A CERTAIN GROUP OF CASES *

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The experimental and clinical work of Homen, Marinesco, T. Buzzard, Russel, Strumpell, Gowers and many others has been of importance in placing the clinical picture of acute disseminated myelitis on its correct pathologic and etiologic basis. It is not the object of this paper to discuss, from either the pathologic or the clinical standpoint, acute disseminated myelitis as such, but to study acute ascending myelitis, an anatomic variation of the same disease. As a means of comparison we have chosen a case of acute ascending poliomyelitis presenting both typical clinical manifestations and pathologic evidence of this disease. Both cases occurred on the service of Dr. F. W. Peabody, in the medical wards of the Peter Bent Brigham Hospital, and with his consent we summarize the cases.

REPORT OF CASES

Case 1.—*History.*—F. T. C., woman, aged 28, an American housewife, who complained of stiffness of the neck, gave a negative family and marital history. There had been no pregnancies. The patient was moderate in her diet and had a fair appetite. The bowels were regular. She had had chicken pox, mumps and measles in childhood. At 14 years she was in bed three months with a complaint of general weakness, diagnosed tuberculosis of the right lung apex. Later she was informed that the process was arrested. Six years before she came under our observation she had had an attack of bilateral otitis media, but had recovered without suppuration or deafness. There had been frequent attacks of tonsillitis in the last four or five years. There was no history of syphilitic infection. Menstruation began at the age of 16. The periods were irregular, coming at intervals of three, four or five months, lasting two weeks and associated with considerable pain. The patient's weight had been 114 pounds two years before; her present weight is 98 pounds. Three weeks previously she had contracted rhinitis, characterized by sneezing, nasal discharge and subjective fever. Six days previously, July 31, 1920, she had developed pain in the pit of the stomach and the back. The following day four teeth were extracted. On August 2, she complained of neck soreness, and the following day she developed fever and stiffness of the neck muscles, with headache and insomnia. The next morning there was pain localized in the back and retention of urine. The day previous to admission she vomited several times and her neck became painful to flexion, retention of urine continued and both legs became paralyzed rather suddenly.

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* From the medical clinic and pathologic laboratory of the Peter Bent Brigham Hospital, Boston, Mass.

Physical Examination.—The patient was a thin, sick appearing woman, mentally clear, lying in bed with head somewhat hyperextended. She was cooperative, but was apparently suffering considerable pain. Her apparent weakness was augmented by the pain on movement of head and body. The skull was symmetrical; the scalp clean. The pupils were regular and equal, and reacted to light and in accommodation. There were no external ocular palsies. The sclerae and conjunctivae were normal. There was no nystagmus. The optic nerve heads and retinas showed no abnormalities. The hearing was equally good in both ears. There was no discharge from the meatus; and no obstruction or discharge from either nostril. The lips were of good color and showed no herpes. There was no disturbance of the muscles of facial expression. The teeth were in fair condition. Several upper and lower teeth had been recently extracted but reaction was slight around the gums. The tongue protruded in the midline without tremor. The tonsils were not enlarged. The palate contracted symmetrically and the gag reflex was present. The voice had not changed in quality and no difficulty was experienced in swallowing. The neck was somewhat extended. Turning of the chin to the side caused pain.

There were no athetoid or choreiform movements of the arms. Fibrillary tremors were not noted in either arm, and there was no atrophy. Strength was equal in both arms and hands, and there was no dysmetria. All forms of sensation were normal. Biceps, triceps and radial periosteal reflexes were neither diminished nor exaggerated, and were equal. There was a flaccid paralysis of both legs save for a slight abduction movement at the hips. The movements of both feet were impaired. Neither leg could be raised from the bed; flexion at the knees was weak but extension at the knee was absent. Both legs showed fibrillary tremors. They lay absolutely flaccid and both knee jerks were absent. The tibialis anticus and the peroneal muscular reflexes were absent on both sides. The Achilles reflexes were hyperactive. There was no Babinski sign, and all forms of sensation were unimpaired. A bilateral Kernig's sign of slight degree was present.

Course of the Disease.—The following day all the voluntary movements of the lower limbs were abolished and the legs lay absolutely flaccid. The Achilles jerks were absent; sensation was retained; and Kernig's sign was lost. Catheterization for relief of retention was necessary. During the course of the day the intercostal muscles ceased to act and the abdominal reflexes were absent. All the trunk muscles were paralyzed and breathing was purely diaphragmatic. The arms were held in a flexed position. On the afternoon of the same day, the left arm assumed an extended position with loss of voluntary motion and abolition of the deep reflexes. Shortly afterward the right arm was in an identical position and similarly showed signs of a flaccid palsy. There were no sensory disturbances. All deep reflexes were absent.

On admission the temperature was 101.5 F., pulse 104, respirations twenty per minute. On the following day (day of death) her temperature was 101 F., pulse 100, respirations 30, with a terminal rise of temperature to 102.4 F., a stationary pulse rate and respirations 40.

Clinical Pathology.—The urine (catheterized specimen) was amber; alkaline; with a specific gravity of 1.022; albumin, the slightest possible trace; sugar, none; sediment, no casts, and no red or white blood cells.

The blood examination revealed hemoglobin 90; white cells 12,800; 85 per cent. polymorphonuclears; 15 per cent. mononuclear leukocytes.

The cerebrospinal fluid, on admission, showed 246 cells, 95 per cent of which were lymphocytes, 5 per cent. polymorphonuclear leukocytes; globulin ++. Fehling's solution was reduced. There were no bacteria seen in the smear. Cultures were made and remained sterile.

A fibrin web[1] formed over night. No acid-fast organisms were seen in stained smears. The blood Wassermann reaction was negative.

Outcome.—The case, then, was one of a young woman mentally clear and alert, absolutely paralyzed from the neck caudad. The accessory muscles of respiration were prominent and overacting. The sternomastoids, alae nasi and the diaphragm all showed strikingly their increased responsibility. The patient realized her distress. She requested that the covers be removed from her abdomen and spoke only in monosyllables. The respirations became irregular and the patient died that evening of bilateral diaphragmatic involvement. There were no cranial nerve palsies as far as could be determined. Her mental status was unimpaired to the end. The duration of the illness was six days.

Necropsy Findings.—Necropsy was performed twelve hours postmortem. The diagnosis was acute anterior poliomyelitis, acute splenitis, and healed pulmonary tuberculosis (right apex).

The body was that of a well developed, fairly well nourished woman. The left axillary gland was a trifle enlarged. The mesenteric lymph nodes were enlarged, moist and reddened on section. Examination of the heart revealed numerous small, red, petechial hemorrhages beneath the pericardium. Otherwise it was not remarkable. The valves and endocardium were normal; the myocardium appeared slightly paler than normal. The lungs were negative except for congestion of the dependent parts. They had a normal pink color, a "downy" crepitant feel and were voluminous in the anterior portions. At the apex of the right lung was a small, opaque, depressed scar. On cutting, the surface of the dependent parts was smooth, and by pressure a large amount of dark, reddish fluid was expressed. The bronchial lymph nodes were swollen and congested. The spleen weighed 130 gm. The cut surface was dark red and of a somewhat soft consistence. Examination of the gastro-intestinal tract indicated that the stomach and large and small intestines were evidently normal, with the exception of a few Peyer's patches which were congested. The weight of the liver was 1,315 gm. It appeared to be normal. The kidneys and suprarenal glands were normal.

In examination of the skull, on removing the calvarium, the dura appeared normal and no fluid escaped. The sinuses contained a few small red clots but no thrombi. The middle ears were normal. Examination of the brain revealed a moderate degree of injection of the pial vessels and a slight diffuse reddening of the gray and white matter throughout. It was not, however, a wet brain. Sections of the cerebrum, cerebellum, medulla, pons and basal ganglions showed no definite lesion. Examination of the spinal cord revealed the superficial veins much distended and the whole cord, particularly in the lumbar and thoracic regions, swollen. Sections taken from various levels showed in the lumbar cord marked softening of the gray matter, particularly of the anterior horns, so that they bulged from the cut surface, in some places being hemorrhagic and so soft that a cavity was easily formed by slightly scraping the surface with a knife.

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[1] Fibrin web formation in poliomyelitis in spinal fluid standing over night as mentioned by Dubois, Am. J. Obst. **74**:1074, 1916, is not an unusual event.

The vessels of the white matter were injected but no definite lesion was discerned there. The degeneration of the anterior horns was marked in the lumbar and lower thoracic regions, but became less in ascending, until, in the cervical segments, only a diffuse reddening and some swelling of the cord was noted. No increased amount of spinal fluid was apparent. A frozen section of the lumbar cord revealed almost entire replacement of the anterior horns by large endothelial (?) cells and polymorphonuclears, very marked vascular dilatation and perivascular mononuclear infiltration. An emulsion of cervical cord in physiologic sodium chlorid solution was made and 5 c.c. injected into the peritoneal cavity of a rabbit. No signs of a reaction of any kind developed during the ensuing four weeks.

Microscopic Examination.—The heart, lungs, kidneys, pancreas and suprarenal glands revealed nothing of importance. In the spleen the pulp sinuses were markedly congested and the malpighian bodies were very small. There was marked hydrops of the liver cells and in the center of the vacuoles there were frequently a few colloid droplets, often the center of a radiating fibrin meshwork. Necrosis of a single liver cell with invasion by polymorphonuclears was not uncommon.

A section of the colon revealed moderate congestion of the mucosal vessels and infiltration of mucosa by mononuclear cells and eosinophils in numbers. Some of these mononuclear cells were endothelial leukocytes filled with hemosiderin pigment. The solitary follicles were hyperplastic, mitotic figures being abundant. Many endothelial cells in mitosis were also present in these areas.

Lymph nodes from the mesentery revealed marked lymph stasis. There were a few small germ centers but as a whole they were atrophic. Most of the cells were small lymphocytes.

Examination of sections from the frontal, temporal, parietal and occipital lobes of the brain revealed congestion of the blood vessels and edema of the perivascular sheaths. In some sections also a moderate number of polymorphonuclear cells were present, diffusely scattered in the white matter, and rarely one was seen attacking a spider cell, the nucleus of which had undergone chromatolysis. The cerebellum was normal. Sections from the level of the fifth, sixth, seventh and eighth cranial nerves showed extensive lesions of the nerve cells and blood vessels. No nuclei had entirely escaped damage and the lesions varied from loss of cytoplasmic granulation and pyknosis of nuclei, to necrosis with phagocytosis by polymorphonuclear neutrophils and endothelial cells. Minute hemorrhages from congested capillaries were found here and there in nuclei and pyramidal fiber tracts. There were also perivascular edema and infiltration about the blood vessels, both in the cord and in the meninges by polymorphonuclears and lymphocytes. More marked lesions were present in the medulla than in the pons. Few nerve cells were intact and more of these revealed degenerative changes in nucleus and cytoplasm. The gray matter was disorganized by the tremendous invasion of polymorphonuclear leukocytes and large neuronophages which had replaced degenerated cells in the nucleus gracilis, radicis spinalis trigemini, and the dorsal nucleus of the tenth nerve. The white matter also manifested some change, the axons of many nerve fibers in funiculi gracilis, cuneatus and fasiculus anterior proprius having disappeared and vacuoles being left in their places. The blood vessels of the meninges, including those of the anterior and posterior median fissures, were dilated and surrounded by polymorphonuclears and endothelial leukocytes in large numbers. There was no dilatation of the canalis centralis. Proceeding cephalad in the medulla the lesions were well marked but the olivary nucleus manifested less involvement than the other nuclei. The damage was always symmetrical and there was little to choose between the various levels as to which indicated the older lesion, but the higher levels were undoubtedly less affected than the lower. No reparative changes were seen except the phagocytosis of necrotic nerve cells.

Examination of the spinal cord revealed striking and extensive lesions in the gray matter at all levels. It was difficult to say where the earliest lesion had been. In the sacral cord it was as marked as in the lumbar with slightly less horn involvement in the mid-dorsal region than elsewhere. There was congestion of the vessels and edema of the meninges and cord with many recent hemorrhages into the gray and white matter. An intact ganglion cell was rare. Necrosis of ganglion cells and neuronophagia was conspicuous at all levels with infiltration of anterior and posterior horns by endothelial cells and polymorphonuclear neutrophils. Perivascular mononuclear infiltration was to be seen about practically every vessel in the meninges and cord. There was acute degeneration of fibers in the lateral, posterior and anterior tracts, although this damage to the white matter was less in the mid-dorsal level than above or below it. There was also edema and mononuclear infiltration of the afferent nerve roots. No organisms were found by Goodpasture's method.

This was a typical case of rapidly ascending acute anterior poliomyelitis.

Case 2.—*History.*—G. S. F., aged 39, American, a salesman, complained of "pain in the back and paralysis of the legs."[2] The family history was negative. The patient had been married five years; his wife was healthy, and she had had one healthy child and one miscarriage. The patient had never drunk to excess. His appetite was good; the bowels regular. He had had measles, mumps and diphtheria as a child, and an arthritic attack four years before. This was not typical of rheumatic fever, however, and there were no sequelae. He had had severe tonsillitis and laryngitis two years before. No venereal history was obtained. He had suffered a fracture of the left leg and subsequent bone plate repair three years before.

There had been no loss of weight up to the present illness. This had begun acutely Monday, June 1, 1920, with severe headache and pain in the lower part of the back. The same evening he became somewhat delirious with a temperature of 101.5 F. The headache continued the next day. The patient was clear mentally. A doctor was called who suspected meningeal involvement. Tuesday night the patient was again delirious with a temperature of 102 F. He vomited without nausea. Wednesday morning he was entirely helpless from the hips distally, save for slight movement of the right leg. Urine was retained, necessitating catheterization. The headache continued and the patient was at times delirious. He was admitted to the hospital the same day.

Physical Examination.—This revealed a well developed and well-nourished white man, mentally somewhat clouded, assuming by preference a prone position. The skull was symmetrical and showed no tenderness. The scalp was clean. There were no scars. The facies suggested a febrile condition. There were no seventh nerve palsies. The pupils reacted rather sluggishly to light but were regular, equal and reacted in accommodation. There were no external ocular palsies. There was slight ptosis of the left lid, but no exophthalmos.

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[2] The history was obtained from the wife and the sister.

There was some sclerosis of the retinal vessels and a definite blurring of the lower part of the left disk. The ears revealed no deafness to superficial tests. There were no signs of motor fifth nerve involvement. The lips were of good color. The teeth were in fair condition with a few cavities noticeable. The tonsils were red but not enlarged and showed no exudate. There were no mucous patches in the mouth. The palate was symmetrical and the gag reflex was present. The tongue protruded in the midline without tremor. The voice was soft; no hoarseness, dysarthria or nasal element was noted. Examination of the vocal cords was not attempted. The neck was slightly stiff but the movements were unimpaired. There was slight cervical adenopathy in the supraclavicular region.

The thorax was symmetrical. The chest expanded but little on inspiration, the breathing being diaphragmatic. The heart was not enlarged but there was a slight increase in retromanubrial dulness. No thrills were felt. There was a protodiastolic gallop rhythm at the apex and at the pulmonic area a snapping second sound. A loud systolic murmur was heard over the entire precordia with its greatest intensity over the pulmonic area. No diastolic murmurs were heard. The radial pulses were equal and synchronous, the rate rapid and the pulse not well sustained. The walls were palpable. The blood pressure was: systolic, 175; diastolic, 30. Examination of the lungs revealed a slight relative dulness over the left back with prolonged expiration but no râles. The right lung was negative. The abdomen was protuberant but symmetrical. No abnormal tenderness, spasm or masses were noted. Respiratory movements were entirely abdominal in character. The abdominal reflexes were not obtained.

The arms were held in the flexed position and could not be raised above the head. The muscular power in the hands and arms was impaired. Dysmetria was noted in the performance of movements of precision. There were no involuntary movements or fibrillary tremors. There was no wasting. Faradic and galvanic tests were not attempted. There was some loss of muscle tone. The legs revealed paralysis of the absolutely flaccid type. Passive movement was in no way resisted, gravity determining their position when support was removed. There were no involuntary movements, fibrillary tremors or atrophy. The knee jerks and Achilles' tendon reflexes were abolished. Kernig's sign was absent.

The sensations of pain, temperature and touch were markedly impaired from the level of the sixth rib distally. Over the left leg there was a slight appreciation of tactile sense but this was indefinite. Deep sensibility was impaired in both extremities.

The patient did not complain of pain.

Course of the Disease.—On the following day, June 4, paralysis had ascended as indicated by the following note: "Patient is semicomatose, irrational and unable to cooperate. Breathing is rapid and diaphragmatic in type. There is flaccid paralysis of both arms and legs." It is unfortunate that no note was made concerning the muscles of the neck.

In the afternoon, the patient's respirations were more shallow, and bulbar symptoms developed. Speech became drawling in character, the tongue was protruded with difficulty and swallowing caused regurgitation into the nose, and choking. Apparently rational and irrational periods alternated until the patient sank into coma from which he did not arouse. Death occurred the morning of the third day from respiratory failure. The duration of the disease was five days. The temperature on admission had been 103 F., pulse rate 100, respirations twenty-three per minute. On the following day the temperature was 106 F., with a relatively slow pulse rate of 102, respirations 26. There was a terminal rise of temperature the morning of death, reaching 109 F. per rectum, pulse 106, respirations 15, becoming irregular. The chart thus showed an involvement of the vital centers, i.e., extreme temperature, rapid pulse and slow, irregular respirations.

Clinical Pathology.—The urine (catheterized specimen) on admission was amber; acid; specific gravity 1.020; no albumin; no sugar; sediment, many white blood cells, numerous spermatozoa, and a few hyaline casts.

The urine (examined on the following day) was orange yellow; alkaline; specific gravity 1.022; no albumin; no sugar; a few white blood cells, no red blood cells, and no casts.

The examination of the blood (on admission) revealed hemoglobin 90 per cent., white blood cells 15,000, 80 per cent. polymorphonuclears, 16 per cent. lymphocytes, and 4 per cent. mononuclear cells.

The spinal fluid (on admission 30 c.c. fluid removed) contained 387 cells per cubic millimeter, 60 per cent. of which were polymorphonuclear, 40 per cent. mononuclear cells; globulin +. No organisms were seen in the stained specimens.

Cultures were made and the fluid was injected into the peritoneal cavity of a guinea-pig.

On the following day, a second lumbar puncture was made. Twenty c.c. of clear fluid was removed, showing 250 cells per cubic millimeter, 75 per cent. polymorphonuclear cells; globulin + +.

Cultures from the fluid of the previous day showed no growth. No fibrin web was formed in the specimen on standing.

A guinea-pig killed six weeks after inoculation had no evidence of tuberculosis.

The blood culture (on admission) gave no growth on blood serum and dextrose broth during ten days incubation.

The blood Wassermann reaction was negative; the spinal fluid Wassermann reaction was negative with 2 c.c.

Necropsy Findings.—Necropsy was performed eight and one half hours postmortem. The diagnoses were acute diffuse ascending myelitis, bronchopneumonia, and passive congestion of the viscera.

The body was that of a well-nourished and well-developed white adult male. Eyes, ears and nasal passages were negative. The teeth were in fair condition. There were two small calcified lymph nodes in the mesentery. The diaphragm extended to normal limits on both sides. The pleural and pericardial cavity and mediastinum were normal. The heart was normal. The lower lobes of both lungs revealed small patchy areas of bronchopneumonia and much edema. The weight of the spleen was 120 gm. It was of normal size; externally, it was not remarkable. On section, it was of rather beefy color. The pulp was quite soft and somewhat diffluent. The gastro-intestinal tract was normal, as was the pancreas. The liver, which weighed 1,800 gm., was much congested. The kidneys were normal except for moderate congestion. The suprarenal glands were normal.

The dura was normal. The brain weighed 1,750 gm. It was unusually soft and edematous, and there was some flattening of the convolutions. The surface was everywhere injected, particularly over the cerebral hemispheres. On section the cortex was gray and slightly suffused with red. Section through the pons revealed a small hemorrhage just below the lower cerebral peduncles. There was marked injection of the decussation of the pyramids. Coronal section through the brain in the region of the internal capsules revealed nothing remarkable. The venous sinuses contained no thrombi. The middle ears and nasopharynx appeared normal.

Examination of the spinal cord revealed no evidence of increased fluid or clouding of the fluid. The meninges were injected throughout the entire length of the cord. The upper dorsal and cervical regions were soft, edematous and rather doughy. A transverse section revealed the normal markings almost completely obliterated, and distinction between gray and white matter in these regions was difficult, and in places impossible. At the lumbar level, there was softening, but less than higher up, and the markings were fairly well defined. The cord everywhere was congested.

Microscopic Examination.—The lungs, spleen and kidneys revealed nothing of importance.

The spleen showed moderate congestion. The lymphoid centers were small.

Examination of the liver revealed a few focal necroses with invasion by endothelial phagocytes.

The frontal, parietal, temporal and occipital lobes of the brain showed vascular congestion and edema of the cortex and meninges. A moderate number of polymorphonuclear wandering cells were present in the pyramidal zones and rarely a glia cell in mitosis was seen. No degeneration of nerve fibers was shown by Marchi's method.

More marked congestion and edema were observed in the cerebellum than in the cerebrum. An occasional ganglion cell of the nucleus dentatus had undergone degeneration as shown by retraction of the axon, chromatolysis of nucleus and loss of cytoplasmic granulation. No neuronophagia, however, was seen nor any Marchi degeneration.

Examination of the pons in addition to much edema and congestion, showed a few small, recent hemorrhages, found at many levels. Scattered rather diffusely through the tissue were nongranular polymorphonuclear cells and many cells of this same type with mitotic figures. Marchi's method revealed acute degeneration of varying amount in a few fibers of the pyramidal tracts and in a small number of the spinal roots of the fifth nerve and median fillet. Perivascular mononuclear infiltration was present in the gray matter and the pia. No neuronophagia had occurred although now and then a ganglion cell had lost its cytoplasmic granules and dendrites.

The same lesions found in the pons were present but were more widespread and pronounced in the medulla. Beneath the floor of the fourth ventricle, proliferation of neuroglia cells and degeneration of the nerve cells of the nucleus ambiguous had occurred, and a few necrotic ganglion cells were invaded by phagocytes. Most of the blood vessels were surrounded by collars of small round cells. Proceeding caudad the lesions in the gray matter became less pronounced with the white matter showing more minute hemorrhages and acute degeneration of myelin sheaths and axons.

The most marked lesions of the spinal cord were in the white matter. Acute degeneration of the myelin sheaths and axons had occurred at all levels but was more marked in the cervical and lumbar levels. This was brought out strikingly by Marchi's method, but was also noticeable with eosin and methylene blue staining. There was no predilection for any special tracts, the fibers in the posterior, lateral and anterior pyramidal tracts being involved. No reparative gliosis had taken place. The sacral and thoracic regions disclosed slightly less myelin degeneration. At all levels was found a perivascular mononuclear reaction in both gray and white matter and in the pia mater. The anterior horn cells showed comparatively little. There was no actual neuronophagia seen below the medulla, although many cells appeared shrunken and had lost their cytoplasmic granules. Many nuclei were swollen, others were pyknotic or had undergone karyolysis. Nongranular polymorphonuclear cells were present in small numbers in both gray and white matter but did not appear to be active in phagocytosis. There were minute recent hemorrhages in the white matter in the lateral pyramidal tracts, in the lateral ground bundles of the cervical cord and in corresponding areas in the lumbar cord. A few hemorrhages were also present in Clark's columns and in the anterior cornua. In the upper cervical cord slight dilatation of the canalis centralis had occurred.

The dorsal and ventral radices disclosed very little cellular reaction and this only beneath the epineurium. But acute degeneration of many fibers was seen in Marchi preparations and occurred in both dorsal and ventral roots in equal amounts. At the cervical level some fibers had completely degenerated; others revealed slight changes. About half seemed to have escaped altogether. No ganglions were available for study. No organisms could be demonstrated in sections of the cord from various levels stained with methylene blue, by Goodpasture's or by Levaditi's method. The lesions in the cord were, as a whole, quite marked at all levels. The sacral segments had suffered as much as the lumbar and it was difficult to determine which was affected first. The thoracic and cervical levels, however, were less involved than the lumbar and sacral.

DISCUSSION OF THE TWO CASES

For comparison we have presented two cases, clinically similar in some respects, unlike in others.

Case 1 approaches the classic picture of acute poliomyelitis of the ascending type. It is that of a young woman, who, apparently in good health, was attacked more or less suddenly with pain in the back, followed by soreness and stiffness of the neck, and by headache and insomnia. Subsequently there developed vomiting, retention of urine and a flaccid paralysis of the lower extremities without sensory disturbances. Anatomically, the virus probably caused early and extensive destruction in the gray matter of the lumbar region, subsequently ascending and descending; for it will be recalled that the knee jerks were absent on admission, as were also the tibialis muscle responses, while the Achilles jerks were hyperactive, only to be lost the following day in conjunction with a complete paralysis of the small muscles of the foot, the muscles of the calf, and the ham string muscles. We are thus correct in assuming an extension of the process, at least to the second sacral segment.

There is abundant evidence of an ascent of the process in the anterior horns. On the day following admission, the abdominal reflexes were abolished and the trunk and intercostal muscles were paralyzed, with consequent diaphragmatic breathing. The arms, as stated, were held in flexion. Theoretically, at least, this sign might be interpreted as a measure of the muscle power of the flexors versus the extensors, the latter being the weaker. Segmentally, one would expect, in an ascending palsy, that the motor cells of the seventh and sixth cervical ventral roots would be attacked at a slightly earlier stage than the motor cells of the sixth and fifth cervical radices. The presence of equally active triceps and biceps reflexes, however, makes difficult such a nice differentiation.

After some hours, both arms became completely paralyzed with loss of tendon reflexes. The signs indicate, therefore, that the lesion had reached the fifth cervical segment with remarkable rapidity. From this stage until the end, it was a matter of but a few hours, the virus attacking the fourth segment, possibly the third, and death resulting from bilateral phrenic nerve palsy.

Her struggle for life was classical and follows so closely a description given by Peabody, Draper and Dochez [3], that we might as well quote from their monograph: "Instead of the normal smooth rhythmic cycle there may be sharp forcible movements of the abdominal wall on expiration. Pressure over the abdomen causes distress and interference with breathing. The alae nasi dilate with inspiration and the accessory muscles of respiration in the neck begin to come into play. As the diaphragm weakens the neck muscles become more and more prominent until it seems as if the whole work of breathing depended on them. The sternomastoids pull on the clavicle and in one case they caused a partial dislocation of the sternoclavicular joint with each inspiratory effort. The head is thrown back and with every breath the lower jaw is pushed forward and downward in a gasping attempt to get air." Such was the terminal picture of Case 1.

The fibrin web formation in the spinal fluid is exceptional but does occur in poliomyelitis.[1] The fluid otherwise is in accord with the meningeal involvement of poliomyelitis, a constant accompaniment of the disease.

The pathologic picture confirms the clinical diagnosis.

Our purpose is not to discuss acute ascending poliomyelitis, the symptoms and pathology of which are known and for the most part well recognized, but as previously stated, to study Case 2, one of acute ascending myelitis, and to offer an analogy between them, perhaps so far as to suggest the same etiology in a small group with this disease complex. This is not an original idea. Recently there have been a few cases of transverse myelitis reported as poliomyelitis of the transverse type. We shall refer to this question later. Suffice it to say, however, that acute myelitis, ascending and descending, is ordinarily classified by most authors as infectious, toxi-infectious, or toxic in

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3. Peabody, Draper and Dochez: Monographs of the Rockefeller Institute for Medical Research **70**:98, No. 4, 1912.

origin and that the streptococcus, staphylococcus, pneumococcus, anthrax bacillus, typhoid bacillus, etc., have been held, and rightly so, as the causative micro-organisms.

A clear and descriptive picture of this complex is quoted from F. Buzzard:[4]

> Acute Ascending Myelitis: This forms a morbid entity which can readily be recognized during life. It is characterized by a progressive paralysis of motion and sensation, beginning in the lower extremities and more or less rapidly climbing up the body, segment by segment, until involvement of the respiratory musculature threatens life and not infrequently produces a rapid termination. The extension of the disease to the brain stem is occasionally indicated by the implication of the cranial nerves before death, and the occurrence of optic neuritis has been recorded in nonfatal cases where all other symptoms have been referable to the spinal cord. With the more usual employment of the lumbar puncture the bacteriology is likely to become better known.

The same author states in his Goulstonian Lecture:

> It is a matter of some difficulty to choose between the terms acute ascending myelitis and acute disseminated myelitis, because the one is probably better supported from the clinical, and the other more appropriate from the pathological point of view, but I choose to include the word "ascending" for the reason that it graphically suggests the prominent characteristics of the disease. Acute ascending meningo-myelitis would do equally well and is in most cases more correct. Reference to the meningitis is, however, no more necessary here than is reference to the pleurisy which undoubtedly complicates the great majority of cases of pneumonia.

In glancing back over Case 2 the clinical picture corresponds fairly well, and the diagnosis can, therefore, be considered as falling in this group. To recapitulate: A previously strong, healthy male adult was taken acutely sick with severe headache, pain in the lower back, fever and nocturnal delirium. The following day vomiting was an added symptom and the third day found the patient paralyzed from the hips down, save for slight movement in the right leg. There was retention of urine. The same day (day of admission) there was noted, in addition, a slightly stiff neck and paralysis of the muscles of the trunk with a resulting absolute diaphragmatic type of respiration. Further, and of great importance, there was marked impairment of all forms of sensation from the level of the sixth rib caudad. Thus, from our anatomic and physiologic knowledge, one must assume a diffuse lesion of both the gray and white matter of the cord, if we are able to exclude acute polyneuritis. The flaccid paralysis and the absent tendon reflexes imply damage of the anterior horns; the loss of the sense of pain and temperature implies damage in the lateral columns, and the loss of deep sensibility designates posterior column involvement. Clinically, then, the process is diffuse and the subsequent course proved it, in addition, to be of an ascending type.

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[4] Buzzard: Quoted in Osler and McCrae's Modern Medicine **5**:*227*.

The next day both arms were involved in a flaccid paralysis. It is unfortunate that the patient was irrational the last twenty-four hours, as more accurate determinations of sensibility, especially of the arms, could have been made. Death occurred with definite symptoms of a brain stem lesion.

This case has been grouped under the caption, acute infectious myelitis, because of a suspected infectious etiology. It can, under clinical grouping, be designated acute ascending myelitis or more appropriately, acute diffuse ascending myelitis. "Myelitis" is here used in the strict sense—inflammation of the spinal marrow. It is more a matter of the degree of inflammatory reaction whether or not certain pathologic specimens shall be classified as an infectious or toxic myelitis. Infective myelitis is a vague term in the absence of septicemia, pleocytic spinal fluid or demonstratable infectious foci. The vagueness of the term is augmented by the absence of demonstrable pathogenic organisms, by culture or spinal cord preparations. Toxi-infectious myelitis, on the other hand, is inapplicable in the absence of foci or infection or convalescence from acute infectious disease.

SIMILAR CASES IN THE LITERATURE

The authors of various texts agree on an infectious etiology of acute myelitis (Oppenheim,[5] F. Buzzard,[4] Bing[6] and many others). Such has been proved many times, both experimentally and clinically, and the literature contains many authentic cases.

Cushman's case[7] was one of acute ascending myelitis in the course of typhoid fever and he was able to demonstrate *B. typhosus* in the cord. Schiff, on the other hand, reports a case of typhoid fever whose cord symptoms began at the end of the first week of the disease. He was unable to demonstrate organisms in the cord (quoted by Lépine).[8]

Localized infection in distant parts of the body needs consideration here. That at times such a focus will give rise to symptoms referable to the cord is well recognized. Whether or not the organisms themselves intermittently gain access to the blood stream and by that route localize in the nervous tissue, or whether there is a selective action of their toxins on the cord, is at present difficult to conclude. Homen[9] has shown that the cocci tend to disappear quickly from the substance of the spinal cord after they stimulate an inflammatory reaction. Negative cultures then prove nothing.

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[5] Oppenheim: A Textbook of Nervous Diseases, trans. by Bruce, Edinburgh, Otto Schulzes & Co. **1**:315, 1911.

[6] Bing: Nervous Diseases, trans. by Allen, New York, Rebman Co., 1915, p. 158.

[7] Cushman: Verhandl. d. Cong. f. inn. Med., 1886.

[8] Lépine: Rev. de méd. **23**:931, 1903.

Recently Olitsky and Kligler[10] have demonstrated an exotoxin or neurotoxin in *Bacillus dysenteriae* Shiga, producing in rabbits, by intravenous injection, lesions in the gray matter of the medulla and cervical cord consisting of necrosis, hemorrhage, neuron degeneration and perivascular mononuclear reaction.

Orr and Rows,[11] instead of injecting organisms or their toxins into the peripheral nerves, placed a celloidin capsule containing a broth culture of the organisms in contact with the nerve. In their first experiments, they used different kinds of bacteria, but later worked with *Staphylococcus aureus* only.

From their pathologic researches, they conclude that "in the spinal and cranial nerves there is an ascending lymph stream to the central nervous system whose main current lies in the spaces of the perineural sheath. Toxins reach the brain and cord by this route and although they spread to some degree in the lymph spaces of the pia arachnoid and so affect structures at a distance from the point of entrance, they pass for the most part in the main stream along the nerve roots into the substance of the central nervous system."

In the examination of their pathologic specimens all the signs of an ascending perineuritis were apparent, extending from the infected focus to the cord which showed all the lesions of a meningo-myelitis. Beside experimental data, these investigators have shown proof by clinical cases and histopathologic preparations.

With these anatomic considerations, given a theoretical case of myelitis which develops subsequent to an ascending infection along the nerves, it seems more logical to expect myelitis dominating the picture, associated with a secondary meningitis, than the reverse.

Some cases of acute myelitis have shown, either by means of spinal fluid or at necropsy, inflammatory reactions of the meninges. It is probably a constant finding in cases due to the localization of bacteria in the substance of the cord. On the other hand, signs of meningeal inflammation may not be so marked in toxic myelitis of bacterial origin. This in contradistinction to experimental evidence seems the only logical assumption when the spinal fluid shows no inflammatory products. As an example of a well-marked localization of organisms in both the cord proper and the spinal meninges, we refer to the case of acute ascending meningo-myelitis with bacteriologic examination, reported by T. Buz-

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9. Homen: Arb. a. d. path. Inst. d. Univ. Helsingfors, 1902.
10. Olitsky and Kligler: J. Exper. M. **31**:19 (Jan.) 1920.
11. Orr and Rows: Brain **36**:271, 1913.

zard and Russell.[12] The cord was soft and congested, showing hemorrhages sufficiently large to be obvious to the unaided eye and more in the gray than in the white matter. The pia arachnoid was milky in appearance over the cord, but over the brain it was normal. Microscopically, in some places the destruction was so great that there was little possibility of distinguishing gray and white matter. In some parts there were enormous numbers of leukocytes and little nerve tissue. All the vessels showed diffuse infiltration of small round cells. There was isolated from the exudate on the meninges and the substance of the cord a gram-negative diplococcus.

For means of comparison, we will refer again to the case reported by Hunt.[13] This patient was suffering from otitis media and a mastoid operation was performed for the complicating mastoiditis. Four days later the patient developed the first symptoms of meningo-myelitis. The picture rapidly developed into an acute disseminated myelitis, but the spinal fluid on two occasions was normal. The first puncture was made a month after the symptoms began.

There were two, or perhaps three, possibilities to explain normal spinal fluid in this connection: first, that bacterial toxins were the causative factor in the myelitis without an accompanying meningitis; second, organisms which had localized in the cord quickly died out, a not uncommon phenomenon, as investigated and noted by Homen. (Marinesco [14] has offered this as an explanation in Case 5 of his series), or third, a spinal puncture might have given positive evidences of an inflammatory reaction at an earlier date.

The world war afforded opportunity for the study of ascending neuritis with subsequent myelitis, but the condition was certainly a rarity. We have not been able to find a typical case. Even the clinical conception of ascending neuritis rests on a very doubtful pathologic basis. The cases, reported by Walshe,[15] of local paralysis following superficial injuries do not seem to be in that category.

Paraplegia urinariae is still unexplained. It is conceivable that an ascending neuritis along the peripheral nerves of the bladder with a localized myelitis may be the important factor concerned, as Leyden has assumed. Walker's [16] three cases of acute ascending paralysis of a most malignant nature had a marked cystitis of long duration. No necropsies were obtained.

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[12] Buzzard and Russell: Clin. Soc. Trans., London **31**:185, 1898.

[13] Hunt: Boston M. & S. J. **174**:788 (June 1) 1916.

[14] Marinesco: Nouv. iconog. de la Salpêtrière **13**:561, 1900.

[15] Walshe: Brain **42**:338, Pt. 4, 1919.

[16] Walker: Clin. Soc. Trans., London **38**:80.

The causative organism in cases of acute meningo-myelitis is suggested by a positive blood culture during the illness, or the determination of inflammatory foci. Evidences of inflammatory reaction in the spinal fluid and positive cultures from fluid taken during the course of the disease are decisive. Postmortem evidence is best obtained by special bacterial stains or by culture from the substance of a pathologic cord. It is to be remembered, however, that the micro-organisms are short lived in the spinal cord. Of debatable importance are those instances in which bacteria in small numbers and of less well recognized pathogenicity are cultivated, either immediately antemortem (twenty-four hours) or postmortem, from the spinal fluid and blood. Such was the difficulty encountered by F. Buzzard [17] in a case of acute ascending myelitis.

It is well to keep these considerations in mind, especially in cases of toxic or degenerative ascending myelitis, the destroyed tissues of the cord acting as a locus minoris resistentiae for an agonal invasion of organisms by the hematogenic route.

It is not necessary here to enter into the much discussed field of Landry's paralysis and acute polyneuritis. It is enough to state that we will probably never know the nature of the disease which called forth Landry's original description of classes of cases which clinically showed a rapidly spreading motor paralysis, affecting first the legs and, ascending, affecting the muscles of the trunk and arms, and producing death by paralysis of respiration. There were no atrophies, sphincteric disturbances or objective sensory findings, fever was slight, and he was unable to detail pathologic changes by the methods at his disposal. It is probably enough to state that such a class of patients still exists which, with modern necropsy technic, shows slight changes in the brain and cord (F. Buzzard,[17] Gardinier,[18] Hun,[19] Stafford.[20])

The etiology of such cases has been much discussed without any uniformity of opinion. The syndrome has been observed in both toxic states (Barker)[21] and infective states (Buzzard).[22] Associated with a toxic state are the class of cases referred to as acute degenerative myelitis. In this condition more evidences of damage are observed in the cord, especially of a degenerative nature, with complete absence of signs of inflammation (Globus).[23]

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17. Buzzard: Brain **30**:65, 1907.

18. Gardinier: J. Nerv. & Ment. Dis. **41**:640, 1914.

19. Hun: New York M. J. **53**:609, 1891.

20. Stafford: Lancet **7**:1172, 1915.

21. Barker: Contributions to the Science of Medicine, Baltimore, The Johns Hopkins Press, 1900, p. 913.

22. Buzzard: Brain **26**:94, 1903; ibid. **30**:77, 1907.

23. Globus: Ascending Toxic Myelitis, J. A. M. A. **69**:816 (Sept. 8) 1917.

Patients with a progressive polyneuritis may present symptoms of an ascending type of paralysis but clinically differentiated from the complex of Landry by pain along the course of the nerve trunks, sensory and sphincteric disturbances. These cases present marked inflammatory and degenerative changes of the peripheral nerves and are correctly classified as acute toxic or infectious polyneuritis (Hunt).[13]

Wickman[24] showed that the fatal cases of poliomyelitis ended with the same clinical picture as Landry's paralysis and that many cases reported with myelitic changes were acute poliomyelitis.

With these remarks on the allied condition of acute ascending myelitis, we now return to the point at issue in Case 2. The prodromal symptoms, fever and leukocytosis, suggest an acute infection. They are not dissimilar to those in Case 1. The negative blood culture is of importance but not conclusive in ruling out a septicemia. There were no signs of a suppurating process, internally or externally, which might have been classed as a primary focus.

The evidence of an inflammatory reaction in the spinal fluid suggested a meningeal involvement, but the causative organism was not demonstrable in smears from the fluid sediment and the cultures remained sterile. Furthermore, no organisms were seen in specially stained preparations from the cord sections. If, as Buzzard has suggested, acute ascending meningo-myelitis is a more correct pathologic term, and surely there is evidence enough in this case of the meningeal element, why then do we fail to find the causative micro-organism? Is it because, as Homen has shown, the organisms tend to die out quickly in the pathologic cord, or as Strumpell,[25] Marinesco and many others have suggested, the process is hemotoxic or lymphotoxic in origin? Lastly, are we here dealing with an ultramicroscopic organism?

As was previously stated, the fact that the virus of poliomyelitis can under exceptional conditions cause more extensive lesions in the cord than are commonly described is not a novel idea. Taylor[26] years ago in discussing a case reported by Putnam,[27] which showed inflammation and degeneration of the gray and white matter of the cord, questioned the diagnosis. He concluded that the case could with difficulty be put in the category of acute poliomyelitis.

Batten and Holmes[28] in material from three cases of poliomyelitis showed that there was degeneration of the proprio-spinal fibers in the ventrolateral columns of the cord. This was not an attempt to prove that the virus of poliomyelitis attacks the white matter of the cord, but an endeavor to trace the endogenous fibers of the cord by the knowledge that poliomyelitis produces its destructive effects chiefly on the gray matter, with resulting secondary degeneration. In the severely affected regions, there were degenerated fibers scattered diffusely throughout the ventrolateral columns, and thus no definite systematization could be recognized.

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[24] Wickman: Nervous and Mental Diseases, Monograph Series **16**:61, 1913.
[25] Strumpell: Neurol. Centralbl. **20**:415, 1901.
[26] Taylor: J. Nerv. & Ment. Dis. **29**:448, 1902.
[27] Putnam: J. Nerv. & Ment. Dis. **10**:14, 1883.
[28] Batten and Holmes: Brain **35**:259, 1912.

Harbitz and Scheel [29] report a case of poliomyelitis showing a diffuse inflammation of the cord and medulla with a more acute, swollen and softened hemorrhagic focus localized in the region of the tenth and eleventh dorsal segments and, microscopically, intense inflammatory reactions in both the gray and white substance. They believe their case represents a transitional form between acute myelitis and poliomyelitis, and that certain cases of acute myelitis should pathogenetically and etiologically be placed in a class with acute poliomyelitis.

Skoog [30] reports a case of poliomyelitis of the transverse myelitis type. The clinical condition suggested a transverse lesion in the dorsal region and the patient died four months after the onset of the paralysis with increasing bed sores, cystitis and other complications. Antemortem cerebrospinal fluid showed a mild pleocytosis with 90 per cent. lymphocytes, very marked fibrin formation, and no organisms. The blood culture was negative. The cord presented marked thickening of the meninges and the lower five dorsal and lumbar segments showed cylindrical microscopic cavities extending up and down in both the white and gray matter, especially marked in the posterior columns. The softening in the sacral and coccygeal region, from which staphylococci were grown, probably did not represent a factor in the primary condition.

There are a few instances reported as poliomyelitis of unusual type and the extent of the process in the white substance of the cord is evident from their symptomatology. Anderson and Frost's [31] case gave symptoms of upper motor neuron damage, i.e., spasticity of the paralyzed limbs, exaggeration of reflexes and no atrophy. The patient's serum protected a monkey from a fatal dose of a known virus according to the method described by Flexner and Lewis.[32]

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[29] Harbitz and Scheel: Epidemic Acute Poliomyelitis in Norway in the Years 1903 to 1906; Results of Anatomic Investigation of Nineteen Cases of Acute Poliomyelitis and Kindred Conditions, J. A. M. A. **49**:1420 (Oct. 26) 1907.

[30] Skoog: Acute Poliomyelitis, Transverse Myelitis Type, J. A. M. A. **59**: 764 (Sept. 7) 1912.

[31] Anderson and Frost: Abortive Cases of Poliomyelitis: An Experimental Demonstration of Specific Immune Bodies in Their Blood Serum, J. A. M. A. **56**:663 (March 4) 1911.

[32] Flexner and Lewis: Experimental Poliomyelitis in Monkeys: Seventh Note: Active Immunization and Passive Serum Protection, J. A. M. A. **54**:1780 (May 28) 1910.

Sach's [33] case presented symptoms of a transverse myelitis in the high dorsal region with retention of urine, almost complete anesthesia from the tip of the xiphoid distally, with paralysis of the lower extremities, increased reflexes, exhaustive bilateral ankle clonus and a positive Babinski's sign. The spinal fluid showed a pleocytosis and the cultures were sterile. Blood and spinal fluid Wassermann tests were negative. Ten days before discharge, her blood serum rendered the virus of poliomyelitis inactive and protected a monkey, thus giving a neutralization test which in most instances is specific. Such a demonstration justified the inference that the patient had passed through the infection of acute poliomyelitis.

At a later date Sachs [34] in a paper on the infectious forms of acute transverse myelitis reports two other cases which were considered by him as poliomyelitis. Both patients recovered. In the same paper he raised the question as to whether we are to suppose that cases of acute ascending paralysis of infectious origin are in any way identical with acute poliomyelitis.

By analogy to a small number of reported cases, by reference to some of the experimental and pathologic data, and by the clinical methods at our disposal, an attempt has been made to show that not all cases of acute infectious myelitis come under the category of a bacterial or bacterial-toxic origin.

There is possibly an isolated group of these cases, clinically presenting symptoms of transverse myelitis and etiologically dependent on the virus of poliomyelitis.

The same possibility is suggested regarding cases of acute ascending myelitis similar to Case 2, here reported. With symptoms of an acute infection, signs of a regular step-like ascent of the process in the cord and bulb, and spinal fluid the character of which is possible with poliomyelitis and necropsy findings which did not aid in establishing a bacterial etiology, we are justified in Case 2 in further considering the cause. The regularity of the ascent is a point against ascending myelitis of bacterial origin as was so correctly demonstrated by Gower's [35] case. The blurring of the left optic disk in our case, a rare phenomenon in poliomyelitis, is not an infallible point of differentiation.[36] The fact that this case immediately preceded a considerable epidemic of poliomyelitis in Boston lends weight to the idea that it was due to a similar infection.

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[33] Sachs: J. Nerv. & Ment. Dis. **39**:747, 1912.

[34] Sachs: Trans. Assn. Am. Phys. **29**:81, 1914.

[35] Gower: Clin. Lect. Dis. Nerv. Syst., 1895.

[36] During July, August and September, 1920, in the medical wards of the Peter Bent Brigham Hospital, five cases of poliomyelitis proved by necropsy findings were observed. In one both optic disks showed diffuse blurring of the margins and haziness of the nerve heads. This rare phenomenon of poliomyelitis is important as regards Case 2, under discussion.

The clinical diagnosis, acute ascending myelitis, means little in these cases, principally because we do not know the cause. Little advancement will be made either in classification or treatment without learning more about etiology.

We regret to state that our evidence is incomplete. Had we pathologic evidence of poliomyelitis after subsequent injection of the cord emulsion into a monkey, our case would be complete. Unfortunately this procedure was not attempted.

As Flexner has remarked, "In my opinion there is no test that is conclusive, pathologically speaking, except the histology of sections prepared from the central nervous system." The pathologic demonstrations complete, therefore, the discussion regarding this case.

CONCLUSIONS FROM PATHOLOGIC EXAMINATION

Case 2 presents many interesting points for study. The pathologic picture agrees with the clinical in that there can be demonstrated an acute ascending affection of the spinal cord with less marked lesions in the basal ganglions and cerebral cortex. Acute myelin degeneration in the cord and spinal radices is the most conspicuous lesion, hemorrhages into the gray and white matter and perivascular lymphocytic reaction are next in prominence, and least marked are degenerative changes in the gray matter and nuclei of the pons. The meningitis present is such as is usually found in acute anterior poliomyelitis and other infectious states involving the central nervous system. The gross and microscopic findings in cord and brain are those of an infectious process, with the virus showing a great predilection for nerve tissue. The vascular lesions are not pathognomonic of any one condition.

Preceding by about a month an outbreak of acute anterior poliomyelitis in Boston and the vicinity, this case necessitates careful scrutiny to remove any doubt as to whether or not it has any relation to the cases of poliomyelitis which followed it. The prominence of the cord lesions would be sufficient to place it in the Heine-Medin group. Degeneration of white matter in poliomyelitis has in practically all epidemics been conspicuous by its absence or insignificance. With a view of determining whether this lesion was duplicated in the epidemic of July and August, the cords of several cases were studied by Marchi's method. These were fulminating cases, the entire illness lasting from three to six days. In one of them as much damage was found in the white matter as was found in the case under discussion.

The diagnosis of choice is acute diffuse ascending myelitis. While it is impossible to be sure that in this case we are dealing with the result of an infection by the poliomyelitis virus, it is equally difficult to deny possibility in view of the clinical picture, pathologic findings (although quite different in order of prominence of the lesions from poliomyelitis), and its occurrence so little in advance of a sizable epidemic of acute anterior poliomyelitis. Also the idea of "change in type of disease," recently emphasized by Sir Humphrey Rolleston, surely must be as applicable philosophy in this disease as in others.

OSSIFICATION OF THE MENINGES *

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"Death seems to be the only object of hope and consolation in all such deplorable cases, as the unhappy patient must be frequently subject to pains, spasms, and occasional dereliction of the faculties, both of body and mind, which . . . are likely to terminate . . . in palsy, mania, idiotism or other maladies more grievous than death itself." Such was the comment of Dr. J. Smith [1] in a letter, written in 1780, to William Hunter, referring to ossification of the meninges, which he had discovered, postmortem, in a recent case. In that period of medical practice, such a pathologic finding sufficed to explain the most grotesque and most divergent clinical findings. Paralyses, chorea, epilepsy, a wide range of psychotic manifestations and feeble-mindedness, constitute a few of the maladies to which Dr. Smith referred. Many case reports were published in an attempt to interpret countless symptoms, the true nature of which the medical man of that period could not otherwise explain, because of what we now know to have been insufficient knowledge of anatomic and physiologic relationship.

Again, the accidental discovery of bony tumors, plaques and extensive ossification of the meninges in persons who had certainly died of systemic disease, and who had not previously manifested cerebral symptoms, led to widespread discussion of the subject.

The literature shows an active interest on the part of medical men, especially during the first half of the nineteenth century; but it is most probable that the celebrated lectures of Virchow, insisting on the benign character of these pathologic changes, checked the impetus of pseudoscientific interpretation, and, as a result, interest lagged, and reported cases since 1870 have been comparatively few. Whether the percentage of occurrence has been actually less, or the presence of meningeal ossification has been observed and noted as unimportant is a matter of question. The fact is that it is most difficult to find any mention of the subject in our leading textbooks on neurology and

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* Read at the forty-seventh annual meeting of the American Neurological Association, Atlantic City, N. J., June, 1921.

[1] Smith, J.: The History of an Extraordinary Affection of the Brain, Med. Obs. Soc. Phys., London **6**:180-185, 1784.

pathologic anatomy. Oppenheim mentions it only to state that ossification is common, and may be extensive, even to the point of enveloping the brain, but that it has no clinical significance.

While none of us today has the temerity even to suggest that bony transformation of the meninges may produce the cerebral mischief mentioned, we are impressed with the possible significance of this finding in certain cases. A careful study of the literature has revealed most remarkable and almost unbelievable osseous changes. While, for the most part, the pathologic reports may be accepted as trustworthy, it is evident that the conclusions in many instances were incorrect. Frequently such findings as milky exudates, hemorrhagic areas, "preternatural congestions," areas of softening, free blood and even pus in the ventricles, indicate at once the probability that the ossified areas were only coincidental changes.

But, in the broader aspect, it is almost certain that the location, extent, depth and other characteristics of osteophytic processes described in the early literature would present to us, in isolated instances, definite signs of intracranial disturbance. Today we are quite aware of the manifest symptoms that may arise from a small irritative lesion of the cerebral cortex or the overlying meninges, especially in certain areas. On the other hand, we are aware that extensive infiltration of the brain substance may occur without giving rise to significant symptoms. Hence it may be of interest to summarize briefly the pathologic changes that have been described to date, in the earnest hope that, in the light of our more advanced methods of examination and interpretation, their occasional clinical importance may be noted.

The falx cerebri easily assumes first place as the most common site. The regions of the dura alongside the superior longitudinal sinus, and the tentorium are next in frequency of occurrence. All areas of the convexity, however, may be affected.

The dura is frequently involved alone, or may be the seat of a process which involves the other tunics as well. Again, ossification may have its origin in the pia-arachnoid, leaving the dura free.

The meninges may be the seat of isolated plaques, minute and multiple, or of clusters, irregularly placed or united to form in plates extending anteroposteriorly the length of the brain and laterally over the convexity so that the brain is practically encased in a bony sheath.

The osseous formations may be pyramidal in shape and multiple, and the convolutions are seen to be molded exactly to the opposing surfaces. Solid masses of bone, the size of a hazelnut and larger, may develop from the inner surface of the pia-arachnoid and become embedded in the cortical substance between the convolutions. These have been invariably associated with corresponding changes in the meninges overlying them.

The thickness of the specimens reported varies between a millimeter to a centimeter, perhaps in the same plaque. The outer surfaces are invariably smooth and glistening, while the inner surfaces are always rough and jagged. Stalactite processes are common, and may be so pointed that the outer tunics are pierced and the cortex invaded for a considerable distance. This is a peculiarly significant feature from the standpoint of symptomatology.

The meninges involved usually manifest a congestion of the vessels, and, especially in those cases in which there has been a history of trauma, adhesions are frequently observed.

A theoretical discussion of the relationship of calcification to ossification will not be entered into; nevertheless it is well to note that many of the published cases have demonstrated true bony changes. We know that areas of the meninges or brain substance that have become calcified through the medium of extravasated blood due to trauma or from any other cause may become ossified. We know that in the presence of osteoblasts such changes may take place in other parts of the body, such as the heart, kidney, pleura and eyeball. Whether such metaplasia in the brain and its coverings is made more readily possible because of the embryologic functions of the dura, or whether through a specific action on connective tissue cells calcium leads to ossification, are questions apart from the purpose of this communication.

Despite the absence of postmortem confirmation, the following case, representing extensive ossification of the meninges, with certain evidences of focal irritation or pressure, is worthy of report. To our knowledge, it embodies the first attempt to study, according to our present day methods, the clinical manifestations of such a pathologic process.

REPORT OF A CASE

History.—Mrs. X., aged 49, married, born in the United States, a housewife, came under observation in March, 1921, through the courtesy of Dr. Evan Evans. She complained of attacks of vomiting, severe headaches, insomnia, languor, and lack of endurance. These symptoms had been particularly prominent for nine or ten years. The patient believed that with the exception of the vomiting, her condition had become gradually worse.

Her mother died at 70, having had trigeminal neuralgia, pernicious anemia and some cardiac disturbance. Her father died of "rheumatism" combined with anxiety. Two brothers were well, and her husband and two children were quite well. There was no history of a similar affection in the collateral branches. As a child the patient was never very robust. She was always considered anemic, and between the ages of 12 and 14 had had severe "fainting spells" but no convulsions. She had had all of the usual childhood diseases, including measles in a particularly serious form. Tonsillotomy was performed during adolescence. Menses appeared at the age of 13 and had always been regular, rather profuse and painful. An attempted curettage and cervical

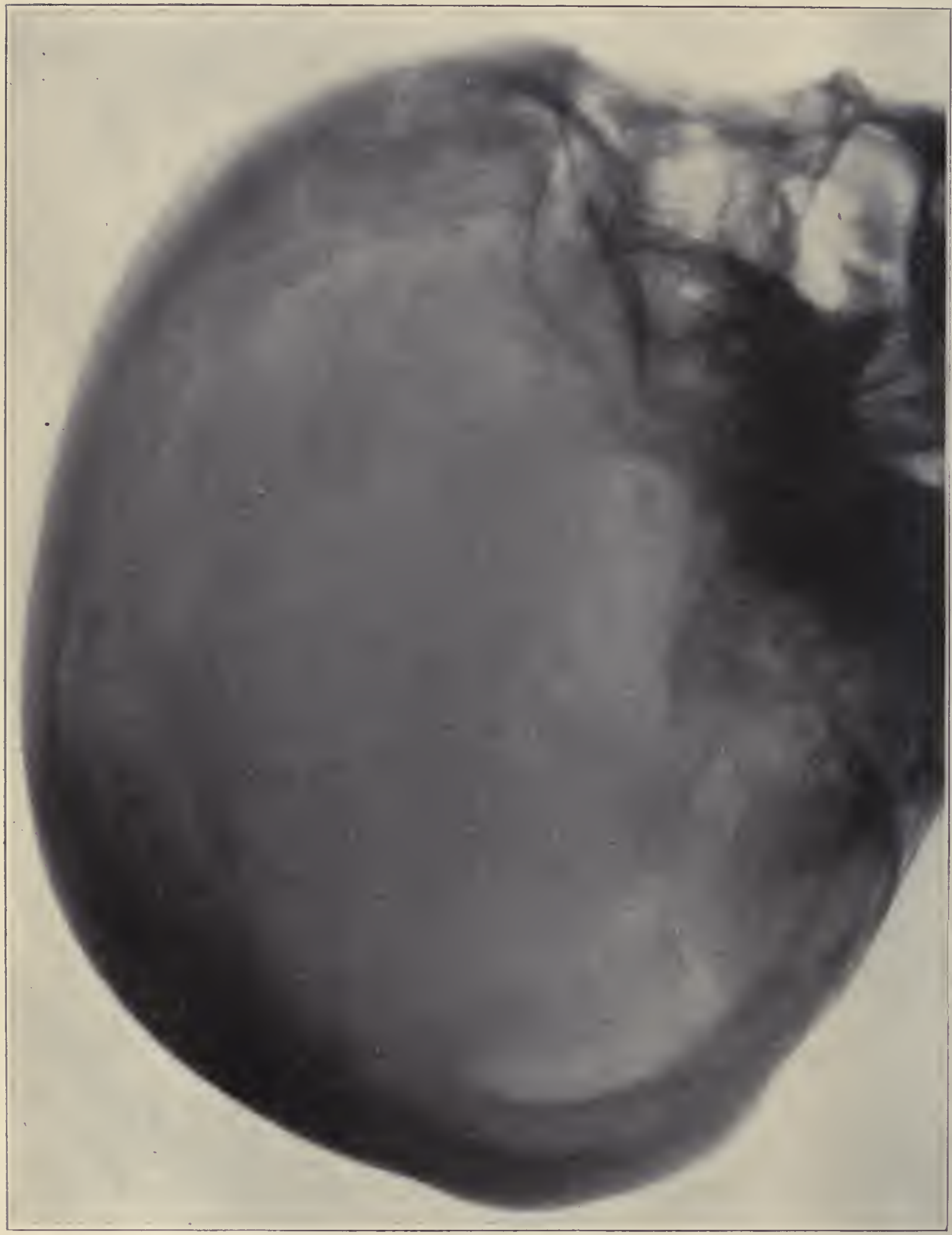

Fig. 1.—Roentgenogram of case reported. Mottling of inner surface of skull which stereoscope indicates is not connected with the base and which is probably due to ossification in the meninges.

repair failed twenty-five years ago because the pulse became so weak under the anesthetic that the surgeon could not continue the operation. There was a vague possibility that a miscarriage occurred twenty-three years ago.

Present Illness.—Since her pregnancies the patient had not been as well as formerly, but during the past ten years especially she had suffered severely with the symptoms enumerated. The care of her mother who died eight years ago had involved a great deal of anxiety and responsibility.

The vomiting spells first occurred about nine years ago at a time when the patient was acutely anxious concerning her son's health. Since that time they had been fairly constantly present, sometimes occurring every day for perhaps two weeks, with free intervals of several weeks occasionally. Three months had been the longest interval during which she had not vomited. Nausea usually preceded the attacks. They occurred during sleep as well as at any time of day. They bore no relation to the taking of food. They were occasionally so severe and prolonged that the patient fell asleep from sheer exhaustion. Walking and exercise in general had repeatedly increased the frequency of her attacks. Excitement had never precipitated them.

The headaches described as neuralgic in type, usually but not always unilateral, had persisted for years. There was no periodicity in their appearance, but they were always present at the menstrual periods as well as at other times. The usual location was frontal, with dull aching and drawing pain sometimes referred to the face and throat. Excitement had frequently precipitated and intensified them. The patient also described "sick headaches" associated with vomiting which she characterized as quite different from the neuralgic type.

Insomnia had existed during the same period of years. Five hours was the average amount of sleep obtained. The lack of endurance and languor had been most troublesome and had led to very much circumscribed activities. They have never yielded to the effects of tonic stimulation.

The general physiologic status was good. The bowels were regular. There was frequency of micturition but never incontinence. Digestion, excluding the vomiting attacks, was normal. There had been no especial change in weight during the past ten years.

Examination.—Abnormal Attitudes and Deformities: There were no abnormal attitudes. There were no deformities, except a webbing of the second and third toes of each foot.

Gait: This was normal in all its phases. Associated movements were normal.

Coordination: Equilibratory: Standing with feet together was done well with eyes open and closed. The patient stood on one foot poorly, right and left.

Nonequilibratory: Finger to nose tests were done well, right and left, with eyes open and closed. Finger to finger, and each finger to thumb tests were normally performed. Heel to knee tests were normal. There was no real adiodokokinesis, but the movements of the left upper extremity were awkward compared with those of the right. There was no dysmetria.

Skilled Acts: There was no apraxia. Handwriting was normal. Speech was entirely normal.

Abnormal Involuntary Movements: There was slight digital tremor on each side, greater on the left than on the right. There were no twitchings, choreiform movements or other subnormalities.

REFLEXES *

Deep †	Right	Left
Pectoral	2 +	2
Biceps	3 +	2
Triceps	3 +	2
Radial	3 +	2
Ulnar	2	2
Wrist	2	2
Suprapatellar	2	2
Patellar	2	2
Achilles	2	2
Ankle clonus	0	0
Superficial		
Ciliospinal	1	1
Supra-umbilical	1	1
Supra-pubic	1	1
Upper lateral abdominal	1	1
Lower lateral abdominal	1	1
Plantar flexion	1	1
Babinski	0	0
Fanning	0	0
Chaddock	0	0
Oppenheim	0	0
Gordon	0	0
Schäfer	0	0

* 0—absent. 1—present. 2—active. 3—very active.

† The deep reflexes have been repeatedly examined on several occasions, and the same inequality has been definitely and invariably found.

Muscle Strength: In all muscle groups muscle strength was much below the average normal. There was, however, no disparity between the two sides that could be determined. The weakness was noticed particularly in the fingers, wrist and forearm muscles, especially in opposed flexion and extension.

Muscle Status: Volume and contour were normal. There was no atrophy or hypertrophy. The consistency was soft and flabby. The tone was normal. There was no perceptible myotatic irritability or myoidema. Electrical tests were not made.

Abnormal Associated Movements: There was a definite Hoffmann sign on the left constantly. It was obtained on the right at times

General Sensory Examination: Touch: Acuity, localization and discrimination were normal throughout. Pain: Superficial and deep pain were normal. Temperature: Moderate degrees of heat and cold were accurately determined. Vibration: Vibratory sensation was normal in the upper and lower extremities. Muscle-tendon: The sensibility was normal in the toes, fingers, forearms and arms.

Stereognosis: The size and form of objects were correctly noted.

Cranial Nerves: Optic, Oculomoter, Trochlear and Abducens Nerves and Optic Apparatus: Visual acuity was not tested. The fields were normal to rough tests. The fundi were normal; the pupils were equal, regular, central and moderately dilated. They responded briskly to light and convergence. There was no strabismus. no ptosis, no nystagmus, no exophthalmus or enophthalmus.

Acoustic and Ear: Acuity of hearing was normal for a watch tick. Air conduction was better than bone conduction on both sides. The Rinne test was normal.

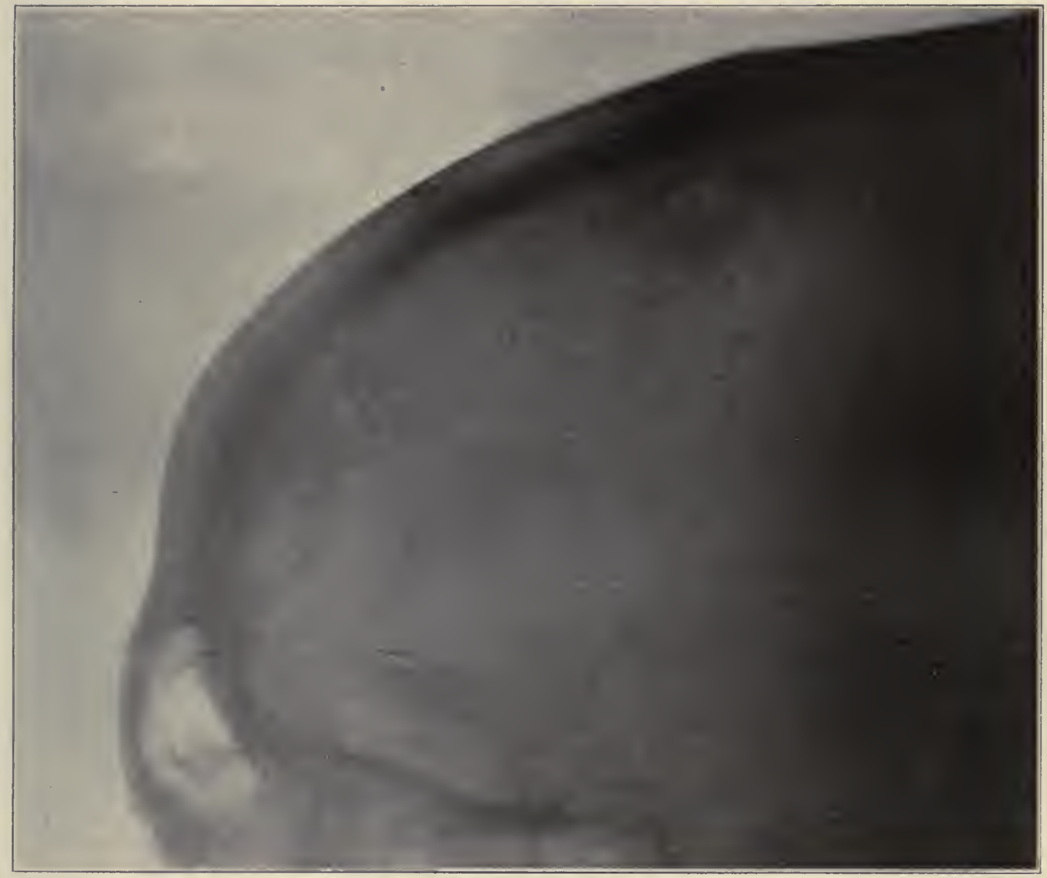

Fig. 2.—Case not accessible to clinical study showing similar osseous changes. Areas of meningeal density which are probably due to ossification.

Trigeminus and Mouth: The motor fifth nerve was normal. The corneal reflex was brisk on each side. There were no abnormalities of the mouth or of the teeth worthy of note.

Facial Nerve and Face: In conversation there was a marked weakness of the lower two thirds of the left side of the face. In smiling this weakness was still apparent. In forced movements, however, such as showing the teeth, and in hearty laughter the weakness was no longer manifested.

Articulation of the labials was normal. Associated movements of the face, eyes and head were normal.

Glossopharyngeus, Vagus Nerves, Pharynx and Larynx: Taste was not examined, but was probably normal. The uvula was normal in position and rose in midline to phonation. Palatal and pharyngeal reflexes were normal. Phonation and swallowing were normal.

Spinal Accessory Nerve: No abnormality in the function of the trapezii or sterno-cleido-mastoids could be elicited.

Hypoglossal Nerve and Tongue: The tongue protruded in the midline; it moved well in all directions and presented no trophic disturbances.

Mental Status: The patient was of an extremely intelligent type. She was alert, of a genial disposition and decidedly not introspective. Her mental life was one filled with detailed care and management of her family and home affairs. She was not irritable, in the ordinary sense, although the usual disturbances of a large household were frequently a source of annoyance which she did not express externally. There was no emotional instability. In fact, the patient's husband considered her especially well balanced. Attention and memory were not defective. There were no phobias, compulsions, hallucinations or delusions.

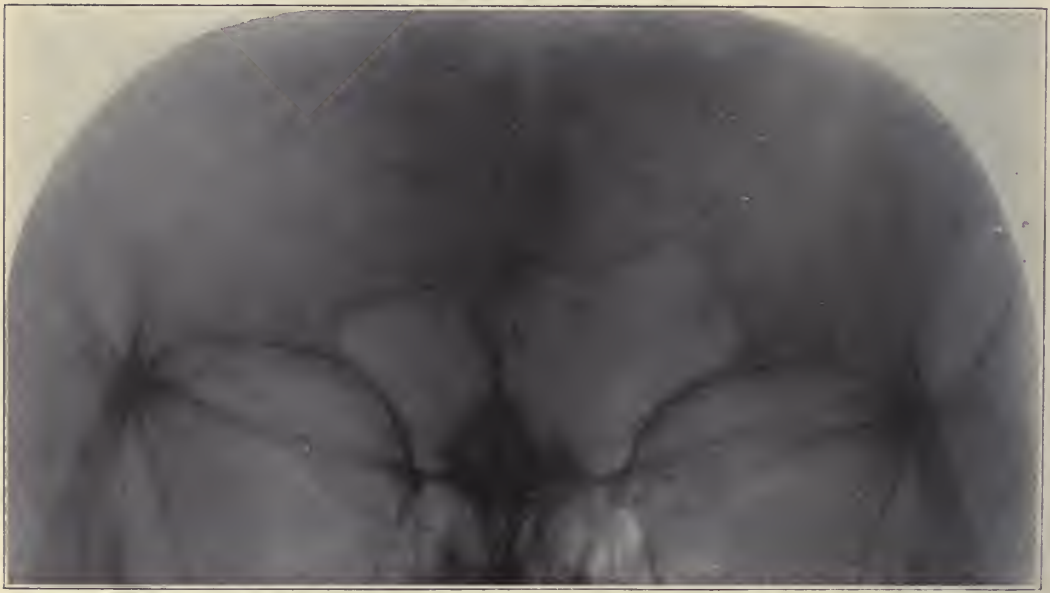

Fig. 3.—Distribution of these ossifications through the frontal areas.

Cranial Morphology: The skull was normal to palpation. There had been no noticeable increase in size.

Systemic Examination: Tegumentary: Her hair was normal in color, amount, texture and distribution. There was no pigmentation of the skin or vasomotor disturbances of any kind. Adipose tissue was rather excessive, but evenly distributed.

Glandular: The thyroid gland was normal. The tonsils were small and almost invisible. There was no palpable adenopathy.

Cardiovascular: The heart was normal. Systolic pressure was 130. The pulse rate was variable, averaging 80.

Respiratory: The lungs were normal.

Gastro-Intestinal: The liver and spleen were not palpable. Palpation and percussion was otherwise normal. (See roentgen-ray report.)

Genito-Urinary: The patient had a rather excessive leukorrhea, but no pelvic signs or symptoms. As stated in the foregoing, micturition was sometimes urgent.

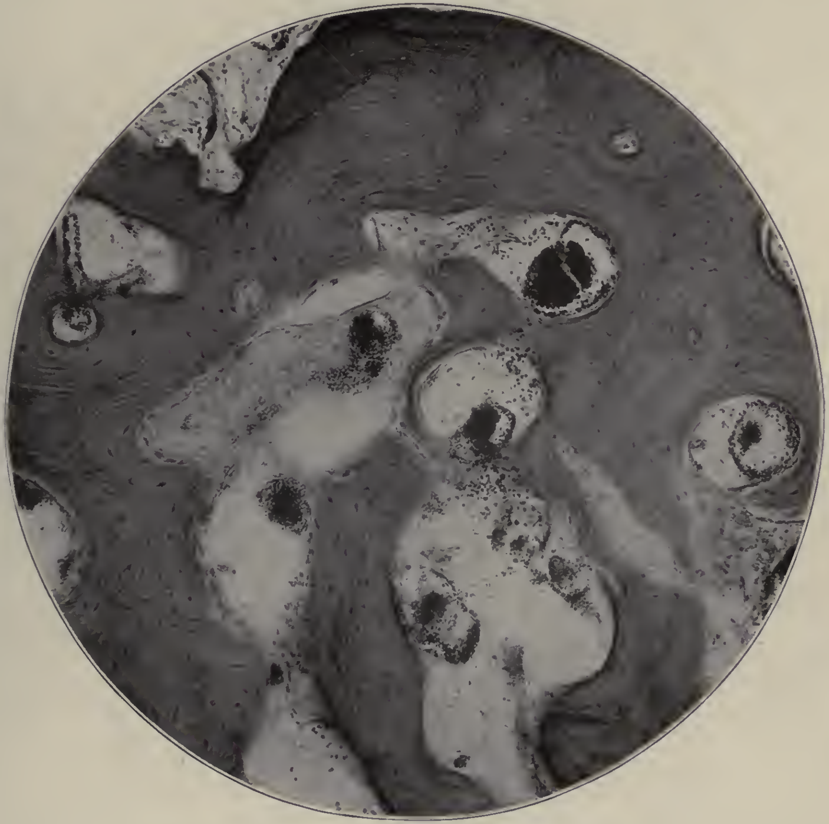

Fig. 4.—Section of osteophyte of the falx cerebri. Specimen submitted by the courtesy of Dr. J. H. Larkin, pathologic department, City Hospital.

Laboratory Examination.—Urine Analysis: The findings were: appearance, very turbid; color, pale amber; reaction, acid; specific gravity, 1.028; albumin, trace; sugar, absent; acetone, absent; diacetic acid, absent; indican, normal; urea, 2.4 per cent.; bile pigments, absent; blood pigments, absent.

Microscopic Examination: The examination revealed: casts, none; cylindroids, none; mucus, a few threads; amorphous, few urates; crystals, none; pus, a few cells; red blood cells, none; bacteria, numerous motile bacilli (contamination); epithelia, few squamous cells.

Blood Wassermann Reaction: The extracts used were crude alcoholic and cholesterinized. The result was negative.

Blood Chemistry: The following determinations were made in 100 c.c. of blood: uric acid, 3.2 mg.; urea nitrogen, 18 mg.; urea, 39.5 mg.; creatinin, 2.6 mg.; sugar, 125 mg. or 0.125 per cent. The examination showed slight nitrogenous retention and hyperglycemia.

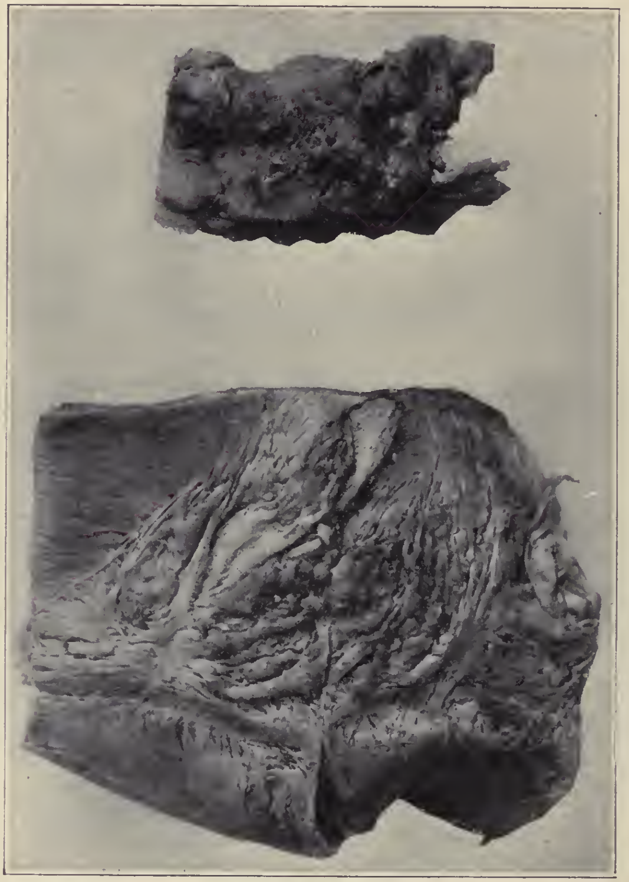

Fig. 5.—Osteophytes of the dura, near superior longitudinal sinus. Note jagged contour of internal surfaces. Microscopic examination revealed true bone. Specimen submitted by courtesy of Dr. Douglas Symmers, Director of Laboratories, Bellevue Hospital.

Complete Blood Count: The blood count revealed: erythrocytes, 4,200,000 per cubic millimeter; leukocytes, 6,000 per cubic millimeter; hemoglobin, 76 per cent.; color index, 0.9 per cent. Differential Leukocyte Count: polymorphonuclear neutrophils, 59 per cent.; small lymphocytes, 38 per cent.; large lymphocytes, 0; large mononuclear leukocytes, 0; transitional leukocytes, 1 per cent.; eosinophils, 2 per cent.; basophils, 0.

The examination revealed a moderate diminution in the percentage of hemoglobin and in the number of red corpuscles, with a fair number of leukocytes and practically a normal differential count.

The red corpuscles were quite uniform in size and shape.

Gastro-Intestinal Serial Roentgen-Ray Taken 1913: Roentgenograms showed residue after six hours. The pylorus was apparently filled out properly. The cecum was rather low, the stomach high.

Skull: Roentgen-ray examination of the skull showed it to be of normal size with a slight increase in its thickness and density. The outer surface seemed to be quite normal, but the inner surface of the frontal and parietal regions was definitely roughened. Stereoscopic plates as well as the anterior ones showed that this was not confined to the longitudinal sinus but was distributed over the entire frontal bone and over an irregular area in the parietal. The stereoscope would further indicate that these processes were of about the same density as the bone and that they projected into the cavity. The sella was not disturbed, and there were no indications of increased intracranial pressure.

Summary of Essential Findings.—These were: history of headaches over a period of nine years; vomiting, occurring at any time of night or day at irregular intervals, aggravated by exercise; pathologically increased deep reflexes of the right upper extremity and bilateral Hoffmann's sign; facial weakness of the central type on the left; roentgenogram of the skull revealed extensive ossification of the meninges.

In a careful and prolonged study of the patient we have been convinced that the signs and symptoms described in the foregoing are due directly to the pathologic condition of the meninges. All other abnormal changes in the skull that might simulate meningeal ossification, including syphilis, carcinoma, tuberculosis and Paget's disease have been easily eliminated.

While our belief that meningeal ossification can be diagnosed during life has not yet been substantiated by postmortem confirmation in this case, it is our conviction that careful investigation in this direction will prove ultimately the occasional clinical importance of this phenomenon.

DISCUSSION

Dr. Beverley R. Tucker of Richmond said that he thought this presentation was of great interest, and he briefly reported a case of ossification of the spinal meninges.

About four years ago he had seen the patient and made a diagnosis of spinal tumor, although the central level was quite indefinite. Her pain, however, was severe and radiated around her body. She had a spastic paraplegia which seemed to be progressive. Her symptoms dated back for about a year. A radiograph of the whole spinal region failed to show anything unusual. Dr. Tucker supposed that it was possibly harder to pick up plaques of this kind on the cord meninges than it was on the brain. Dr. Stuart McGuire of Richmond operated on the patient, and, much to their surprise, found ossified plaques—some of them separate and some overlapping. They were a little larger than the little finger nail; however, some were larger and some were smaller. They exposed an area of about three and a half vertebrae, and although ten of these plaques were removed, they felt that they had not removed all of them. The case was closed up and the patient had been under his observation ever since. He thought the interesting part of it was that the spastic paraplegia had progressed and seemed to have extended up the cord. The spinal cord in the region of most of the plaques had dwindled to about half of its size, showing atrophy; it took on a conelike shape. The specimens of these plaques were sent to the laboratory and were reported as cartilage or bone.

It had been about four years since the operation and one arm was considerably involved by pain and some disturbance of motion, and the other arm was slightly involved. This woman had never shown any cerebral symptoms, as far as they could elicit. He had hesitated to advise operation but had a surgeon examine her with a view to the possibility of another operation to remove more of these plaques, but the prognosis seemed so uncertain that nothing had been done.

Dr. Ernest Sachs of St. Louis said that these ossifications were particularly interesting to him because of the great difficulty of differentiating endothelioma, which are known not infrequently to come from the dura and which not infrequently present a picture similar to those which are seen with ossification, and he would be very much interested to know whether Dr. McKendree could throw some light on how to differentiate these ossifications from ossified endothelioma.

He had operated on two patients, in both of whom he and Dr. Moore, the otologist, believed that there was more of a tumor than the calcification in the dura.

Dr. Charles A. Elsberg of New York said that the Association ought to be much indebted to Dr. McKendree for having brought forward this interesting subject. He had been interested and often much worried because he had seen so many ossifications and calcifications in the meninges and had not understood their significance. He had asked himself whether they could give rise to symptoms.

In many instances of degenerative disease of the central nervous system, and especially in atrophic spinal cords, these calcified plaques often exist in large numbers. There may or may not be a history or the evidence of trauma. The question was whether these calcifications were not entirely secondary to the disease of the nervous system and whether they could give rise to symptoms or influence the changes in the diseased nerve tissue.

Dr. Charles A. McKendree of New York, in closing, said that in reading the literature of the subject, he had found ossified and calcified plaques of the spinal meninges mentioned frequently. Also, in the lower animals, especially the dog, progressive spinal paralysis attributed to ossification of the dura and pia-arachnoid had been reported by French veterinarian schools.

In discussing the matter with several prominent pathologists he had learned that they were commonly encountering ossified areas in the spinal membranes, but that these lesions were not very extensive and did not have the characteristics of depth and penetration that the cerebral plaques had. Therefore, he would be surprised, in general, to find that they could be as significant in man as the cerebral ossifications.

With reference to Dr. Sachs' question as to endotheliomas, he would say that in the case reported there were none of the usual signs of increased intracranial pressure. The symptoms had endured for at least ten years, and the findings could not justify the diagnosis of cerebral neoplasm.

OBSERVATIONS ON CHARACTER OF THE ONSET OF SPINAL PARALYSIS WITH REFERENCE TO THE SIGNIFICANCE OF THE APOPLECTIFORM TYPE OF ONSET IN CONTRAST TO THE SLOW PROGRESSIVE DEVELOPMENT OF PARALYSIS *

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I desire to present a few observations on the significance of the variations in the character of the onset of certain forms of spinal paralysis, for I believe that in some instances the nature of the pathologic conditions present may be determined by the mode of onset alone.

Extramedullary spinal tumors are generally found to be either fibromas or endotheliomas. They grow slowly, producing gradually a weakess of the limbs that increases progressively. Spinal thrombosis, on the other hand, is marked by a sudden or apoplectiform onset; it is not a rare condition, and is usually caused by syphilitic arteritis of one or more of the blood vessels of the spinal cord. The sudden onset of spinal paralysis, however, has also been regarded as indicative of hemorrhage within the cord; but spontaneous hemorrhage, or hematomyelia without trauma, is so exceedingly rare as to render its occurrence most doubtful, for it is not probable that normal blood vessels would burst unless they were injured.

Of the cases of spinal tumor recorded here, four were extramedullary fibromas. Early in their growth they gave rise to pain, due to irritation of the sensory nerve roots. Pain may be the first symptom of a pathologic condition and may be present long before the tumor has attained sufficient size to exert pressure on the cord and thus induce motor weakness. In one case (Case 4) pain was complained of five years before motor symptoms were observed; in another (Case 3), pain was present three months before other symptoms developed, whereas in still another (Case 1), the motor and sensory symptoms appeared almost simultaneously.

The prolonged duration of severe unilateral pain as the sole symptom of disease is most difficult of interpretation. It is important to remember that it may be caused by a spinal tumor. This has fre-

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* Read at the Forty-Seventh Annual Meeting of the American Neurological Association, Atlantic City, N. J., June, 1921.

quently been referred to in the literature on the subject, Auerbach[1] having published the description of a case in which this symptom was the only one present.

Some observers assert that the absence of pain is confirmatory evidence that the tumor is an intramedullary, and not an extramedullary, growth. However, in my case (Case 5) of intramedullary tumor—an ependymoma—pain was present, but not until long after motor weakness had developed. Such a distinction, made on this basis alone, is not justifiable, for many instances are recorded of extramedullary tumors that attained considerable size without involving the sensory roots sufficiently to cause pain; this is particularly true if the tumors grow from the anterior surface of the cord. Pearce Bailey[2] has referred to painless tumors of the spinal cord. The statistics of Steinke,[3] however, based on an analysis of 330 cases, show that spinal tumors grow most frequently from the posterior and posterolateral surfaces of the cord.

Elsberg[4] has reported cases of tumor growing from the anterior surface of the cord in which pain was absent. It would seem, therefore, that, at least in the early stages, the presence or absence of pain is dependent on the situation of the tumor.

Marie and Foix[5] have endeavored to show that the distribution of muscular atrophy induced by an intramedullary spinal growth may not be identical with that caused by an extramedullary spinal tumor. In my Case 5 muscular atrophy appeared to be extensive. Despite the importance of these observations, the differential diagnosis of these cases will, in the vast majority of instances, always be extremely uncertain.

Patients who are paralyzed in the lower limbs as the result of tumor of the cord not infrequently complain of severe pain in the legs, in spite of the fact that cutaneous sensation is diminished or even abolished. For example, in Cases 1 and 3 severe pain in the paraplegic limbs was complained of constantly, although there was anesthesia of these parts. Anesthesia dolorosa, on the other hand, caused by irritation of the sensory spinal nerve roots, is a familiar symptom, but is different in character from the painful sensations to which I have referred. Elsberg has mentioned pain in the lower limbs occurring in spinal cord tumor, resembling, in some respects,

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1. Auerbach: J. Psychiat. & Neurol., 1910.
2. Bailey, Pearce: Painless Tumors of the Spinal Cord, J. A. M. A. **63**:6 (July 4) 1914.
3. Steinke, C. R.: J. Nerv. & Ment. Dis. **47**:418 (June) 1918.
4. Elsberg: Arch. Neurol. u. Psychiat., January, 1921, p. 64.
5. Marie and Foix: Rev. neurol., 1914, No. 4, p. 327 (Society Report).

the painful sensations that Förster[6] records in a recent article, and which Nonne, in a discussion of the subject, called a neighborhood symptom, or "fern Symptome." Sargent[7] also speaks of "pain in the legs" caused by tumor of the cervical portion of the cord.

Spontaneous aching pain occurring in regions that are not supplied by the sensory nerves directly affected by the tumor is difficult to explain. It has frequently been suggested, however, that symptoms may arise from certain limitations in the circulation of the spinal fluid caused by occlusions produced by the tumor, together with alterations in the blood and lymph stream within the cord due to pressure; or they may be brought about by disturbances in the vascular supply of the paralyzed limbs themselves, due to loss of vasomotor tone in the peripheral circulation. Holmes[8] has described pain—or central pain, as he calls it—occurring after gunshot wounds, which he attributes in part to swelling and distention of the injured cord due to edema. It is probable that one or all of these factors may explain, at least in part, the occurrence of pain in the lower limbs, even when the tumor is situated on the cervical or on the thoracic portion of the cord.

In most cases of tumor of the cord motor paralysis takes place so gradually that the actual date of its appearance cannot be determined. It generally begins insidiously, with subjective sensations that are, at first, rarely recognized as important, but that are, in reality, the earliest expression of the rapid exhaustion of weakened muscles.

It is the very gradual transition from a normal to an abnormal state that the patient is quite conscious of, but that the examiner is unable to demonstrate as the beginning weakness that constitutes the earliest phase of severe paralysis.

Cases of sudden onset of motor paralysis in spinal tumor must be exceedingly rare, but Förster refers to one case of this kind in his series, although he gives no details.

Müller[9] recorded a case of tumor growing from the upper cervical portion of the spinal cord that had caused a sudden onset of unilateral paralysis and later remission of these symptoms occurred.

In one case of spinal tumor seen by me, but not recorded here, one lower limb that was already moderately weak suddenly became almost completely paralyzed. This might be compared with the sudden onset of paralysis that occurred in two cases of Pott's disease, reported by me,[10] without deformity of the vertebrae, and caused by myelitis due to compression of the cord, in which there was microscopic evidence of thrombosis of the vessels in the spinal cord.

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6. Förster: Deutsch. Ztschr. f. Nervenheilk. **70**:64, 1921.
7. Sargent, P.: Brit. M. J. **1**:37 (Jan.) 1920.
8. Holmes: Osler's Anniversary Volume, 1921.
9. Müller: Deutsch. Ztschr. f. Nervenheilk. **71**:183, 1921.
10. Cadwalader, W. B.: Am. J. Med. Sc. **112**:546, 1911.

Remission of symptoms during the course of tumors of the cord must also be exceedingly rare, although they are not unknown. Thus, Hedinus and Henschen[11] describe a case, confirmed by the necropsy findings, in which there had been a complete remission of motor symptoms for a short time, and Jumentie[12] reported before the Society of Neurology in Paris a most remarkable case of tumor of the cervical portion of the cord that had caused severe paralysis of all four limbs, and in which spontaneous remission had occurred and continued for three years. The case subsequently ran the usual course, and the diagnosis was eventually confirmed by postmortem examination.

No variations in the character of the development of spinal paraplegia could be more strikingly different than is the onset of paralysis in acute softening of the spinal cord caused by obliteration of the spinal vessels. In one (Case 6), paraplegia developed within a few hours; in another (Case 7), paralysis was complete in one hour.

Spiller[13] has recorded a case in which the first symptoms of paralysis developed within fifteen minutes after the man had been lifting heavy blocks of ice, severe paralysis occurring a few minutes afterward. At necropsy thrombosis, with softening in the first thoracic and eighth cervical segments of the cord, was demonstrated. This was attributed to syphilitic infection. A somewhat similar case is reported by Barth and Léri,[14] in which the patient, six months after treatment for syphilis, suddenly developed headache, vomiting and fever; at the end of the second day he was completely paraplegic, and on the third day the right arm became paralyzed and bulbar symptoms appeared. The patient died seventeen days later. Microscopic examination of the spinal cord showed cellular infiltration of the meninges, marked infiltration of the blood vessels, multiple hemorrhages in the anterior horns and obliteration of the central canal. This was, without doubt, a syphilitic process.

Schultze,[15] in a recent article on acute myelitis, points out that syphilitic thrombosis of the spinal vessels is the most important single factor in causing the sudden onset of paraplegia.

Spiller asserts that spinal thrombosis is probably the cause of most of the apoplectiform palsies that occur during the course of myelitis.

The weight of evidence seems to show quite clearly that the sudden onset of spinal paralysis may mark the advent of more than one disease. Perhaps the best known condition in which this occurs is acute epidemic poliomyelitis although other forms of acute myelitis may have a similar onset. In a larger number of so-called cases of acute myelitis, however, the affection is due not to an inflammatory process, but to softening from thrombosis of some of the vessels of the spinal cord, particularly in syphilitic infection, a view that was held years ago by Bastian.[16]

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[11] Hedinus and Henschen: Jahresb. u. d. Leistung auf dem Gebiete Neurol. u. Psychiat. **17**:748, 1913.

[12] Jumentie: Rev. neurol. **28**:285, 1921.

[13] Spiller: J. Ment. & Nerv. Dis., December, 1908.

[14] Barth and Léri: Rev. neurol., 1913-1914, No. 19, p. 393.

[15] Schultze: Deutsch. Ztschr. f. Nervenheilk. **68**:1, 1921.

The belief formerly held by Gowers, that long-continued overexertion of the lower limbs, associated with exposure to cold and wet, as occurred in my Case 6, might be responsible for spinal paralysis, has been voiced again by Schultze. However, experience in pathology and the exceptional opportunities afforded by the late war for observing the effects of such conditions have not disclosed any reliable evidence that I know of to show that these factors alone are capable of producing acute spinal paralysis in men who were in other respects normal. On the other hand, it is quite certain that long-continued, fatiguing exertion under unfavorable conditions may be the causative factors determining the moment of onset of spinal paralysis in persons already diseased, as, for example, in those with latent syphilitic infection of the nervous system or other spinal cord disease.

The rapid or sudden development of ataxia may occur when the posterior column of the spinal cord is diseased, without actual motor paralysis, and if it is very severe, because of its sudden development, it might be confused with actual motor paralysis. Such acute ataxia was seen by me[17] in dogs whose anterolateral tracts of the spinal cord had been cut for experimental purposes. In the report of these experiments the condition was referred to as a pseudoparalytic state.

The following notes are taken from the case history of a man who already had tabes and who suddenly became extremely ataxic:

Robert T., a letter-carrier, was under treatment in the outpatient department of the Orthopedic Hospital in 1911 for tabes dorsalis. He had the usual classic symptoms of that disease of moderate intensity, but there was very little or no ataxia. He could walk well without assistance, and despite advice to the contrary he was still pursuing his regular vocation. Not feeling as well as usual and complaining of his legs being tired, he entered a restaurant to get a cup of coffee, sitting on a high stool at the lunch counter. On getting down from the stool his legs suddenly gave way, and he fell to the floor. He stated that he was unable to walk, and had to be assisted. Subsequent examination in the ward of the hospital, only a few days later, disclosed no additional evidence of tabes or other spinal cord disease beyond strikingly severe incoordination of the lower limbs. He was entirely incapable of walking alone. As tested against the resistance of the examiner, the muscular power of the lower limbs was fairly good. After prolonged treatment, chiefly by rest and diet, and reeducational movements, he was able to walk with the aid of two canes, and though the ataxia persisted, there was no motor paralysis.

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[16] Bastian: Lancet **2**:1531, 1909.

[17] Cadwalader, Williams B., and Sweet, J. E.: Experimental Work on the Function of the Anterolateral Column of the Spinal Cord, J. A. M. A. **58**:1490 (May 18) 1912.

In this case extreme ataxia had at first been mistaken for motor weakness. Without a postmortem examination in this case the actual character and extent of the alterations within the spinal cord must necessarily remain undetermined, the clinical signs indicating only posterior column disease.

In another case coming under my notice the patient, who had been regarded as healthy, during the stress of excessive fatigue and exhaustion incident to warfare rapidly developed severe ataxia, the symptoms of tabes, which had not been previously suspected, having remained in abeyance until this time.

The notes of the following case show that ataxia can develop acutely in the lower limbs in a man suffering from posterolateral sclerosis of the spinal cord associated with severe anemia:

Mr. H. was admitted to a private ward at the Presbyterian Hospital in 1920, and gave the following history:

He had been annoyed for some months by a sensation of numbness in his feet and hands and a general feeling of weakness, particularly after the exertion of walking to his office. At that time, Dec. 15, 1919, he had been examined by his physician, who found that his tendon reflexes were absent. The case was erroneously regarded as one of tabes. About February, 1920, he stated that the numbness in his limbs was increasing, and that his legs felt weak—so much so that he remained at home, although he was still able to walk. On February 9, he was unable to walk, even to take a step, and could not stand erect without assistance, due to extreme incoordination of the lower limbs. On examination, the tendon reflexes were found to be absent; the muscular power of the lower limbs as tested against resistance was remarkably good, but he was unable to recognize the position in which the toes, and even the feet and lower portions of the legs, were placed. Vibratory sensation of the legs and arms was absent. The blood count showed: hemoglobin, 39 per cent.; erythrocytes, 2,440,000; leukocytes, 5 200. Gastric analysis showed an absence of free hydrochloric acid.

The alterations of the cord that are associated with these severe types of anemia, in my opinion,[18] are caused by a toxemia, and are not entirely dependent on the anemia itself, that is, using the term anemia in the sense of indicating only a diminution in the quantity of normal blood.

Such rapid development of extreme ataxia is, in my experience, most uncommon, but these examples are recorded here merely to permit a contrast to be made with true spinal paralysis.[19]

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[18] Penna. M. J., July, 1921, p. 692.

[19] Since the preparation of this paper, an article has been written by Guillain (Bull. de l'Acad de méd., Par. **85**:732 [June 28] 1921), in which he refers to acute ataxia developing in the course of a few hours without any muscular paralysis following an exceptional physical strain in cases of tabes.

Minor (quoted by Oppenheim) states that, of the many cases of hematomyelia observed by him, in only two cases did hemorrhage occur spontaneously. In all the others trauma seemed to be the etiologic factor.

Oppenheim[20] attributes nine-tenths of all cases of hemorrhage to trauma, although he recognizes the fact that in certain affections in which the blood vessels are weakened by disease, for example, in arteriosclerosis, hemophilia and purpura, spontaneous hemorrhage may occur. H. Fabritius[21] reported two cases that came to necropsy in which paralysis associated with fever and other symptoms of acute infection developed suddenly. In the first case he found acute softening of the cervical cord, with thrombosis of the veins and hemorrhage in the anterior median fissure. In the second case hemorrhage took place in the substance of the thoracic portion of the spinal cord, and there were, in addition, perivascular infiltrations, etc. He regarded both as cases of acute myelitis and pointed out that in the second one spontaneous hemorrhage had occurred. These cases seem to show that thrombosis and minute hemorrhages may occur in the myelitis of acute infectious diseases, with rapid development of paralysis. The so-called hemorrhagic type of acute myelitis is generally recognized as the result of an infectious process, but it differs markedly from, and should not be confused with, intraspinal hemorrhage or hematomyelia the result of trauma. Hematomyelia probably never occurs without either trauma or vascular disease, for, as has been stated, it seems most improbable that normal blood vessels could burst unless they were injured or diseased.

REPORT OF CASES

CASE 1.—*Fibroma of the fifth and sixth cervical segments of the spinal cord that had caused gradually increasing weakness of all four extremities. Severe pain in back and in legs. Upper limbs held in the Thorburn position.*

History.—William S. (Lab. No. 563), admitted on March 6, 1911, to the service of Dr. Spiller. The patient's present condition began about November, 1908, with a feeling of heaviness and weakness of the lower extremities. For some time he had had pain in the left upper limb, particularly during the night. These symptoms continued and increased steadily, until five weeks previous to admission, when he was compelled to give up work and remain at home. Recently he had complained of marked pain in the lower limbs on rising in the morning. The weakness of his lower limbs had gradually progressed, and he stated that he was unable to move his legs.

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[20] Oppenheim: Textbook of Nervous Diseases, trans. by Alexander Bruce, 1911.

[21] Fabritius, H.: Zur Kenntnis der akuten Myelitis und der sogenannten spontänen Hämatomyelie, Arb. a. d. pathol. Inst. a. d. Univ. Helsingfors, N. F. **1**:125, 1912.

Physical Examination.—The patient lay with the forearms flexed on the arms so that they were at an acute angle, the hands resting on the chest, just above the breast, in the Thorburn position (Fig. 1). The forearms and hands were wasted to a moderate degree, but the upper arms were not atrophied. Passive extension of the forearm caused pain, probably due to beginning contracture of the biceps tendon. There was complete paralysis of all the muscles of the fingers and hands. Movement was impossible in the fingers and wrists. Extension of the elbow was not possible, owing to complete paralysis of the triceps. Flexion of the forearm at the elbow was possible, but was weak, due to paresis of the biceps, brachialis and brachioradialis. Supination of the forearm was very weak, owing to extreme paresis of the supinator brevis. Adduction of the upper arm was impossible because of paralysis of the pectoral muscles. The deltoid was paretic. The entire inner half of the arm on the extensor and flexor sides was anesthetic, as were also the entire hand, all the fingers and the forearm, except a small radial zone beneath the elbow where there was hypesthesia. The middle of the posterior part of the upper arm over the triceps was anesthetic. The region supplied by the circumflex nerve on the upper arm and shoulder was hypesthetic. The wrist reflexes and biceps tendon reflexes of each side were moderately but equally diminished. The triceps reflexes of each side, however, were much exaggerated, though equal. (Here the notes are incomplete, and the report of the subsequent examination stated that the tendon reflexes were abolished

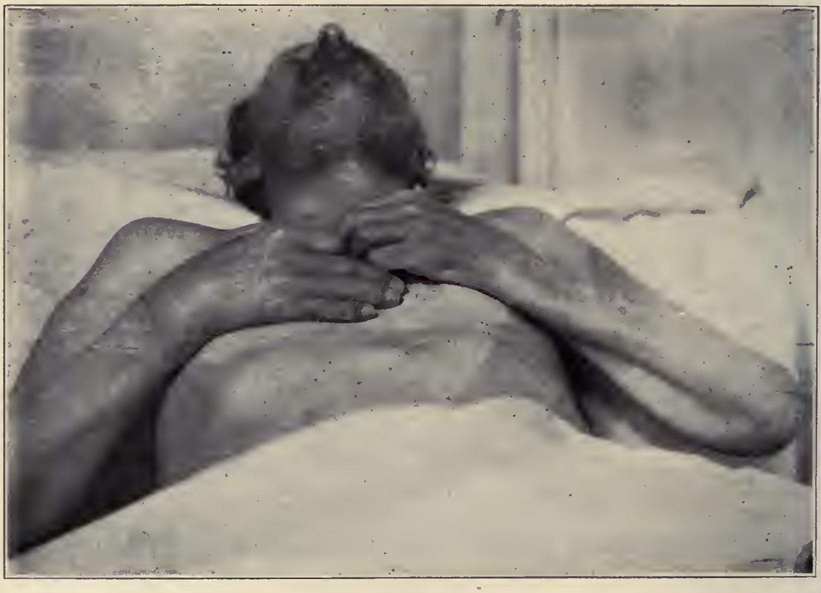

Fig. 1 (Case 1).—The credit for recognition of the significance of this peculiar position of the upper limbs is given to Sir William Thorburn, but he mentions that it had been observed and recorded by others previously. He, however, emphasized the importance of this syndrome as indicative of a lesion above the sixth cervical roots. (Sir William Thorburn: "A Contribution to the Surgery of the Spinal Cord," 1889.)

on each side.) The lower limbs were moderately spastic, but handling, instead of increasing spasticity, seemed to produce flaccidity when the first resistance was overcome. The patellar tendon reflexes and the Achilles tendon reflexes were equal but exaggerated. There was no patellar clonus and no ankle clonus reflex. On irritation of the soles of the feet there was no movement of the toes. The patient had difficulty in controlling the sphincters of the bladder and anus.

Over the trunk tactile sensation was lost to a point as high up as if a line were drawn through the nipples. Sensations of heat, cold and pain were greatly diminished, but not entirely lost. The senses of touch, heat, pain and cold were lost throughout both lower extremities, but he rarely felt a pinprick over the anterior surface of the right thigh.

He complained constantly of severe pain in the lower extremities, and frequently asked the nurse to move his legs.

The Wassermann test of the blood appeared to be negative. Urine analysis showed evidences of cystitis. There was no deformity of the spine. The patient grew gradually weaker and weaker, and died, March 24, 1911, fifteen days after admission.

Postmortem Examination.—A fibromatous tumor was situated between the fifth and sixth cervical segments of the spinal cord. The tumor extended through the fourth cervical and fifth cervical intervertebral foramina, and grew about 2 inches downward into the soft tissues external to the vertebrae. The tumor had grown on the left anterior and left lateral surfaces of the cord, compressing the fifth cervical with the sixth cervical segment. The upper part of the sixth cervical segment was slightly compressed. The left fifth cervical anterior and posterior roots were involved by the tumor.

Examination by the Marchi method disclosed degeneration in one crossed pyramidal tract and in the posterior column of the same side. This was probably due to injury of a spinal root below the level of the lesion—evidently the descending fibers of the fifth and sixth cervical roots. The descending degeneration was surprisingly slight.

CASE 2.—*Fibroma of cervical portion of spinal cord beginning with pain in the right hand, with gradually increasing weakness of right upper limb. Four months later gradually and progressively increasing motor weakness of the left upper and both lower limbs.*

History.—Miss L. E. F., aged 35 (Lab. No. 400). The patient was healthy until 1897, when she first began to complain of numbness and a tingling sensation in the fingers of the right hand, associated with gradually increasing weakness, which caused her to drop objects. Several months later wasting of the muscles of the right hand was noticed. Gradually the whole arm became weak, and the pain extended to the right scapular region. Pain was much more frequent and severe at night than during the day. Several months after the numbness and tingling of the hand were first complained of the right lower limb grew weak, and the left upper limb also became affected. This condition increased steadily, so that after about four months the right lower limb was so weak that she was unable to walk without assistance. The left lower limb soon became similarly affected.

Physical Examination.—This revealed great weakness in the whole right and left upper limbs. The fingers and thumbs were habitually carried in flexion, suggesting the "claw-hand" appearance. The extensors were weaker than the flexors, although the latter had little power. The thenar and interossei muscles of each side were greatly atrophied. The right wrist reflexes were distinctly more marked than the left. Both lower limbs were weak and spastic, but the right was affected more than the left. The tendon reflexes in the lower limbs were both greatly exaggerated, the right more so than the left. Ankle clonus was absent on the left side, but was occasionally present on the right. Sensation was much diminished on the right side. (Notes incomplete.)

Constipation was present, an enema having to be administered every third day. Urine was passed involuntarily at times, but, as a rule, the patient was compelled to use a catheter.

Postmortem Examination.—A fibroma of the cervical region of the spinal cord was found.

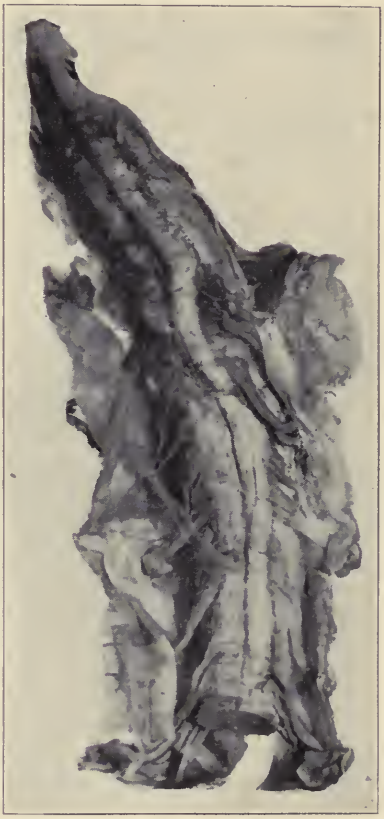

Fig. 2 (Case 1).—Fibroma of the fifth and sixth cervical segments of the spinal cord that had caused gradually increasing weakness of all four extremities; severe pain in back and in legs. Upper limbs held in the Thorburn position.

Case 3.—*Fibroma of cervical portion of spinal cord pressing on the first thoracic, eighth cervical, posterior spinal nerve roots that had caused severe pain in the upper and the lower limbs, followed three months later by slowly developing motor weakness.*

History.—Mrs. A. W. G., aged 43 (Lab. No. 539), was admitted to the service of Dr. Spiller at the University Hospital, Sept. 15, 1909. The patient stated that in October, 1908, she developed severe pain in the right hand. This was diagnosed as neuritis. At this time she also had an extremely tender point in her back, on the left side, between the spinal column and the left scapula. The pain in her right arm continued for about ten weeks, and then gradually subsided and disappeared. Since that time she had had a persistent numb sensation in the fingers of the right hand. For some time past there had been pain in the left arm and numbness of the fingers of the left hand. About two and one-half months later—in December—the patient developed severe pain, limited to the left foot; this lasted about three weeks and then gradually disappeared. It was attributed to gout.

In February, 1909, about four months after the onset, she began to complain of a feeling of numbness about the waist, and of a sensation of distention in the abdomen.

About December, 1909, the patient first noticed that the left leg seemed to "lag" a little in walking, and in March, 1909, there was marked weakness in both lower limbs, although the left was weaker than the right. On March 27, 1909, while getting into a hot bath, she suddenly discovered that there were numbness and loss of sensation in all the parts below the waist. The following day the patient went to bed and remained there. After six weeks the weakness had increased to such an extent that, although just able to stand erect, she was unable to walk. She had now lost control of the bladder and rectum. Shortly after first going to bed she noticed that the legs would contract spasmodically. She stated that "any little thing" caused the muscles of the lower limbs to contract, and at the same time there would also be severe pain in the legs, radiating upward and stopping at the painful point between the shoulders. There was a constant burning sensation in the lower limbs, and the patient had a bed-sore of two or three weeks' duration.

Physical Examination.—The pupils were equal and reacted normally to light in accommodation and convergence. The movements of the eyeballs were normal. The cranial nerves all functioned normally. When the patient was turned to the right while lying down, there was sharp, stinging pain in the left middle finger. The grasp of each hand was normal. Biceps and triceps tendon reflexes were normal on each side, although the triceps reflex of each side may possibly have been less active than the biceps reflexes. Tactile, pain and temperature sensations were normal in each upper limb. No difference in sensation was apparent on the radial and ulnar sides of the limbs. Ataxia was absent, and there was no impairment in the sense of muscular position. Stereognostic perception was normal. The lower limbs were extremely rigid. The thighs were held flexed on the trunk, and the legs were strongly flexed on the thighs and could not be extended by the examiner. The patellar tendon reflexes could not be obtained because of the spasticity. Ankle clonus was present on each side, provided it was tested for when the spasm was partially relaxed. The Babinski sign was typical on each side. Tactile, pain and temperature sensations were lost throughout the lower limbs. There was pronounced involuntary jerking of the lower limbs. No deformity of the vertebrae, even at the point of tenderness in the back, was present. There was complete loss of control of the bladder and rectum.

September 20, an operation was performed by Dr. Frazier. The laminae of the first, second, third and fourth dorsal vertebrae were removed. On nicking the dura, cerebrospinal fluid spurted out for a distance of 2 or 3 inches, evidently under considerable pressure. About 50 c.c. of fluid escaped. After the cord had been fully exposed for a distance, including the first three thoracic vertebrae, nothing abnormal could be seen beyond a suspicious prominence at the level of the first thoracic segment. Because of the patient's condition, further exploration was deemed inadvisable. The wound was closed with sutures. On the day following the operation the patient complained of almost constant severe pain, and died later.

Postmortem Examination. — A well-defined fibromatous tumor, measuring 2.5 by 1.7 cm., was found firmly attached to the anterior surface of the cord. The first left posterior thoracic root lay on top of the tumor, which pressed on and extended to the upper part of the first thoracic segment. The growth touched and pressed on the eighth cervical anterior root, but did not implicate it. The dura was adherent to the tumor in its upper portion. The growth was situated in front of the cord and did not extend to its right edge, but protruded beyond the left edge.

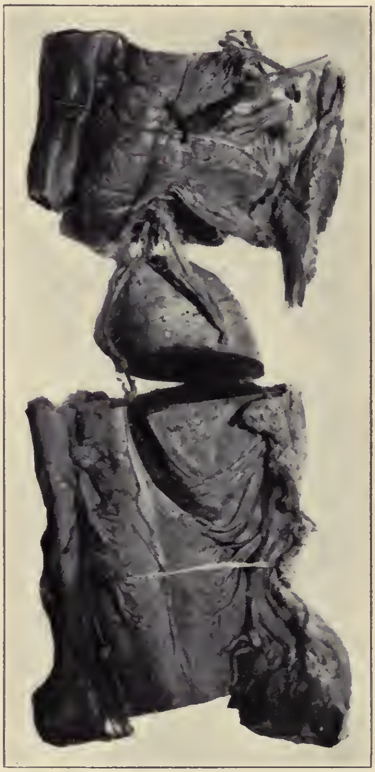

Fig. 3 (Case 2).—Fibroma of cervical portion of spinal cord beginning with pain in the right hand, with gradually increasing weakness of the right upper limb. Four months later gradually and progressively increasing motor weakness of left upper and both lower limbs.

Microscopic Examination. — Examination by the Marchi method showed intense degeneration of the ascending and descending tracts.

Case 4.—*Fibroma of the thoracic portion of the spinal cord that had caused burning pain in the epigastrium for nearly three years before gradual development of spastic paraplegia and anesthesia; successfully removed by operation.*

History.—Fannie P., aged 49, was admitted to the University Hospital in Dr. Spiller's service, Oct. 8, 1920. The patient had entered the hospital in 1915 on account of burning pain in the epigastrium and vomiting, and had left a few weeks later, against the advice of the staff. Pain had been attributed to cardiovascular disease. Examination at this time revealed no symptoms of disease other than evidences of beginning cardiac failure. She was readmitted to the hospital in October, 1920, still complaining of burning pain in the epigastrium, between the shoulders, and extending down the spine. She said the pain was severe at times. Examination showed that the tendon reflexes of the lower extremities were equal and much exaggerated. Ankle clonus was present on both sides. There was a positive Babinski sign on the right side, but none on the left. The spinal fluid showed 5 cells to the cubic millimeter; positive globulin reaction; sugar was present.

November, 1920, the muscular power of the lower limbs against resistance was fairly good for individual movements, but in walking her lower limbs appeared to be weak. The reflexes of the lower extremities were equal and exaggerated. Ankle clonus was distinctly marked on both sides. The Babinski reflex was absent on each side. Bone conduction, tested with the tuning-fork, was normal over both lower limbs. The sense of position in the toes was normal. Touch and pain sensations were normal over the lower limbs. The patient left the hospital before the examination could be completed.

She was readmitted in March, 1921, still complaining of severe burning pain in the back and in the epigastrium. On examination the following findings were noted: an exaggeration of the biceps and triceps reflexes; profound paralysis of both lower limbs, with exaggeration of reflexes, ankle clonus, and marked spasticity. Tactile, pain and temperature sensations were completely abolished below a point corresponding to about 2 inches above the level of the umbilicus. Vibratory sensation and the sense of muscular position were slightly impaired in the lower limbs. Spinal puncture elicited 4 c.c. of yellowish fluid; five cells per cubic millimeter; globulin positive. The Wassermann reaction was negative. Roentgen-ray examination showed no deformity of the spine. The patient suffered considerably from intermittent spasmodic flexor contractions of the lower limbs.

Treatment and Course.—April 12, 1921, an operation was performed by Dr. Frazier. The laminae of the fifth, sixth, seventh and eighth thoracic vertebrae were removed. A subdural tumor occupied the posterior aspect of the cord, extending from the eighth to the eleventh thoracic segments. The tumor extended farther on the left than on the right side, a small protuberance extending from the growth around the lateral aspect of the cord above the middle of the tumor. After the removal of the tumor the spinal cord was found to be very much flattened. Microscopic examination proved it to be a fibroma.

Case 5.—*Intramedullary infiltrative tumor (ependymoma) of cervical portion of spinal cord; duration five years, causing slowly progressive increasing spastic paralysis of the limbs with marked muscular atrophy and increased reflexes and burning pain in the back.*

History.—Joseph M. (Lab. No. 671) was admitted to the University Hospital, Oct. 15, 1914, in the service of Dr. Spiller. In 1909 the patient first noticed weakness of the right arm, without pain. This progressed gradually. Two years later the right leg began to grow weak, and he had a sensation of numbness and tingling in the leg. Still later the left leg became similarly affected, and finally, in 1914, nearly five years after the onset, the left upper limb became paretic.

For the past three years the patient had had considerable burning pain between the shoulders and in the lumbar region, numbness and tingling of the lower extremities, with subacute aching pain in the knees and ankles.

Physical Examination.—This revealed marked kyphosis, beginning in the cervical region and extending into the thoracic region, with the point of greatest curvature at the fifth thoracic vertebrae. The ocular examination was negative. The cranial nerves were normal. The muscles of the upper extremities and shoulder girdles, including the supraspinati and infraspinati, deltoids, pectorals, and all the muscles of the upper arms, forearms and hands, were greatly wasted. The muscles of the thenar and hypothenar eminences were relatively well preserved. Those of the left upper limb were not quite so markedly affected as were those of the right upper limb; the latter was moderately spastic, and there was marked clonus at the wrist joint. Fibrillary tremors were present in the deltoids, biceps and triceps. The biceps and triceps reflexes on the right side were exaggerated. The biceps on the left was exaggerated, but the left biceps reflex was absent. The muscles of the lower limbs were considerably wasted, and there was corresponding weakness. The tendon reflexes were equal but exaggerated, and there was abortive ankle clonus on each side. The Babinski reflex was also present on both sides. Cutaneous sensation of every form was impaired over the arms, trunk and lower limbs. At times the patient seemed to recognize pain, but his answers were uncertain, and exact limits were not determined.

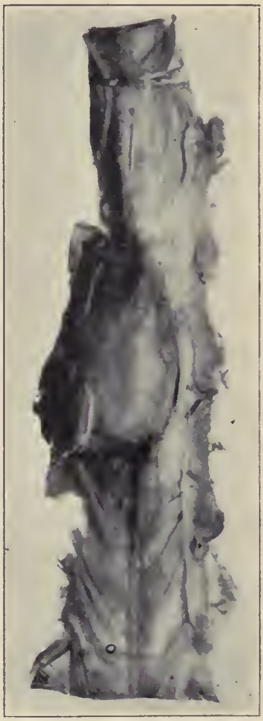

Fig. 4 (Case 3).—Fibroma of cervical portion of spinal cord pressing on the first thoracic, eighth cervical, posterior spinal nerve roots that had caused severe pain in the upper and the lower limbs, followed three months later by slowly developing motor weakness.

Postmortem Examination.—This revealed marked fusiform swelling of the cord, its point of greatest intensity being at the lower cervical and upper thoracic segments. On cross-section, under the microscope, the specimen showed the characteristics of an ependymoma.

Case 6.—*Sudden development of paraplegia caused by acute syphilitic softening of the spinal cord from thrombosis.*

History.—Robert McElreth (Lab. No. 267) was admitted to the Philadelphia Hospital, Dec. 20, 1904, in the service of Dr. Mills. After having been exposed to severe wet and cold weather in November, 1904, the patient was suddenly seized with "chills" and with a feeling of weakness, and was compelled to remain in bed. A few hours later he was unable to move his legs or to control his bladder and rectum. Vomiting occurred frequently.

Physical Examination.—This showed that the pupils were equal, but did not contract to light, although they did contract in accommodation. The cranial nerves were normal, as were also the upper limbs. The feet were swollen, deeply cyanotic and cold, pressure producing deep pitting. Both knee reflexes, although present, were decreased; no patellar reflex or ankle clonus was present, but there appeared to be a suggestion of dorsal flexion of the great toe. On plantar irritation he could move both legs slightly—the right more than the left. The man did not feel the prick of a pin below the knee, but at about that level, and above it, there appeared to be sensations to pain. Although oblivious to light touch on the thighs, he distinguished firm palpation, whereas over the abdomen and elsewhere sensation seemed to be normal.

Treatment and Course.—Dec. 20, 1904, 10 p. m.: The patient vomited, apparently the result of calomel. The bowels did not move. Temperature was lower; there was incontinence of urine.

Dec. 21, 1904: First sound just barely audible, about fifth interspace, in nipple line; second aortic sound could be heard faintly, but second pulmonic sound was not discernible. Breathing was better, easier and slower, and the pulse had become somewhat slower. The bowels opened by incontinence; there was a slight tendency to vomit after the midday meal.

Notes by Dr. McConnell: There was no palsy of any muscle supplied by the seventh or motor fifth nerve. The external ocular muscles were normal. The pupils were somewhat contracted, but equal; the left responded to light and accommodation; the right responded to accommodation but not to light. There was no loss of sensation to touch or pain over the face or scalp, and muscular power in both upper extremities was good. All normal movements were good. No wasting of the arm or forearm was apparent; the thenar eminence of the left side seemed somewhat less sound than on the right; there was no wasting of the interossei, although the first interosseous of the left seemed somewhat wasted compared to the right. Both upper limbs were the seat of numerous tumors. These were irregularly round, freely movable under the skin and not painful. They were somewhat soft, but were not cystic. The reflex was normal in the upper extremities of both sides. No tremor or fibrillary twitchings were present. All forms of sensation seemed to be well preserved.

Dec. 23, 1904: Following an enema the night before, the bowels moved well. The patient did not vomit, was perfectly clear mentally and fairly comfortable. At 9:30 this afternoon he suddenly complained of a sense of suffocation and died immediately.

Case 7.—*Acute syphilitic myelomalacia of the thoracic portion of the spinal cord that had caused sudden onset of complete flaccid paraplegia and incontinence of urine and feces.*

History.—Peter S. (Lab. No. 787) was admitted Nov. 21, 1919, in the service of Dr. Spiller. In April, 1919, the patient first began to have difficulty in emptying the bladder. He was treated in another hospital, from which it was reported that a suprapubic cystotomy was done and the bladder drained. During operation the prostate was observed to be enlarged, but it was not removed. In spite of this difficulty the patient continued at his work until Nov. 11, 1919, when, after returning from work and feeling tired, he lay down on a sofa to rest. One hour later, on attempting to rise, he found that he was unable to do so. He stated that he felt dead from the waist down, and that he was unable to move either lower limb.

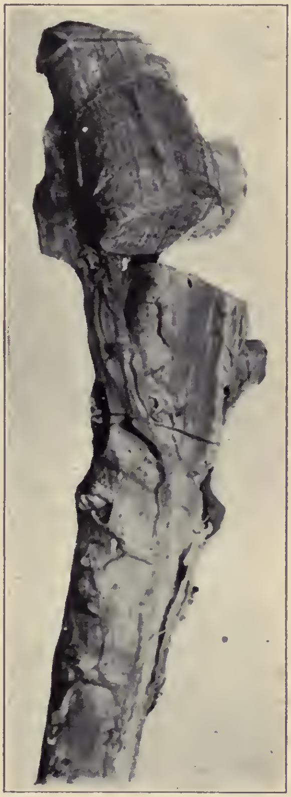

Fig. 5 (Case 4).—Intramedullary infiltrative tumor (ependymoma) of cervical portion of spinal cord; duration five years, causing slowly progressive increasing spastic paralysis of the limbs with marked muscular atrophy and increased reflexes and burning pain in the back.

Physical Examination.—The pupils were equal in size and normal, and reacted normally to light, accommodation and convergence. All cranial nerves functioned normally. There was complete motor and sensory paralysis of all parts below the level of the fifth rib on the left side and below the fourth rib on the right side. Above this level no abnormalities were found. The arms were normal. The lower limbs were completely paralyzed and flaccid. The tendon reflexes were absent on each side, and all forms of sensation were abolished. There was incontinence of urine and feces.

Twenty-five c.c. of spinal fluid were withdrawn for examination and showed 3 lymphocytic cells to the cubic millimeter. The Wassermann reaction was not reported.

Roentgen-ray examination of the spinal column showed evidences of osteoarthritis of the sixth dorsal vertebra and the lumbar vertebrae.

The patient gradually grew weaker. The lungs became congested, and he was delirious at times. The patient died, Dec. 12, 1919.

Postmortem Examination.—Examination revealed complete softening of the spinal cord, involving the fifth, sixth, seventh and eighth thoracic segments. On cross-section it was impossible to differentiate between the white and the gray matter.

Microscopic Examination: The point of greatest intensity of softening was at the level of the fifth thoracic segment. There were thickening of the vessels and perivascular infiltration, but very little meningeal infiltration. Softening was diffuse, though greater in the posterior and lateral portions. Some of the vessels seen on cross-section appeared to be occluded.

Diagnosis: Syphilitic arteritis and meningomyelitis.

ABSTRACT OF DISCUSSION

Dr. Charles A. Elsberg of New York stated that he had always thought that the acute paralysis which occurs within a few hours or within twenty-four hours, as frequently seen in malignant disease of the spine, was due entirely to interference with the circulation of a localized area of the spinal cord and not to an acute myelitis from a toxic cause. When such spinal cords are examined postmortem or perchance are seen on the operating table, no evidence can be found of pressure on the cord, and the outer surface of the cord apparently looks normal.

Dr. Cadwalader deserved much credit for having called attention to the importance of the circulatory disturbances in these acute paralyses of spinal origin.

Dr. Elsberg stated that he hoped in the near future to give an explanation for early sensory pain symptoms and loss of motor power in the lower limbs in disease in the cervical cord. In spite of the evidence to the contrary, which is found in the literature, he believed that there is a lamellar arrangement in the various tracts in the pyramidal as well as in the spinothalamic pathways, and he hoped soon to have the clinical and experimental proof. For example, if it were a fact that in the spinothalamic tracts the fibers for the lower extremities coursed along the outermost parts of the tracts, it could easily be understood that a pressure from the outside would first affect the fibers for the lower extremities. In spite of all the evidence to the contrary, there was considerable evidence that a similar grouping occurred in the pyramidal tracts.

The probable reason that the symptoms which Dr. Cadwalader had described —that is, an affection of the lower extremities first in high lesions—were not more often observed was that a pressure is rarely so slow and gradual that the outermost portions of these pathways are first affected alone. Usually the entire tract is quickly involved.

Dr. Elsberg said that he realized and was fully aware of the fact that, up to the present time, the studies made of ascending and descending degenerations in the spinal cord seemed to show that there is no grouping according to extremities of the fibers in the spinal tracts. But he believed that the evidence had been misinterpreted, and he hoped to be able to bring forward the proof of the correctness of his contention.

Dr. H. H. Hoppe of Cincinnati said that he wanted to add a contribution to the subject of the sudden development of paraplegia in lesions of the spinal cord running a subacute or chronic course.

A patient was admitted to the neurological service at the Cincinnati General Hospital with the history of pains affecting the abdominal region for a few months. Two or three days before admission, she developed weakness of the legs which advanced to a complete flaccid paraplegia in from twenty-four to thirty-six hours. At operation the dura was laid bare and a distinct loss of pulsation showed below the area of exposure. The pathologic condition proved to be a pachymeningitis interna hypertrophica. The lesion was located about the region of the eighth dorsal segment. The sudden development of the flaccid paraplegia in this case was in all probability caused by a compression of the anterior spinal artery.

Dr. William G. Spiller of Philadelphia said he was interested in Dr. Elsberg's remarks and awaited with much interest his presentation of proofs of a dissociation of the motor fibers for the upper and lower limbs in the crossed pyramidal tract of the spinal cord. All investigations, so far as he was aware, had shown that the fibers for the upper limb are not distinct from the fibers for the lower limb in the crossed pyramidal tract of the spinal cord. Experimental work on the motor cortex, in removing the center of the motor fibers of the upper limb or of those of the lower limb, has shown that the degeneration of the crossed pyramidal tract in the spinal cord as determined by the Marchi method is diffuse, and that the motor fibers of either upper or lower limb are not confined to a definite portion of the crossed pyramidal tract. It is doubtful whether such a separation of motor fibers for the limbs exists below the internal capsule, although theoretically one would expect a separation of this kind to exist.

In the posterior columns the fibers from the upper limb are distinct from those from the lower limb, for the column of Goll is believed to consist of fibers entering below the midthoracic region.

Dr. M. Allen Starr of New York said that he did not think all investigations pointed the other way and cited a case with illustrations, which was published in his book in 1902, of some very interesting specimens given him by Dr. Osler, in which a tumor lying opposite the first and second lumbar segments on one side resulted in an ascending degeneration of the posterior columns. This was traced upward at different levels and confirmed the statements that were published twenty-five years ago, that as demonstrated by the experimental lesions studies by the Marchi method there was a lamellar arrangement of fibers as they ascended the cord, each succeeding entering fiber lying on top and posterior to the fiber above it.

Dr. Henry M. Thomas of Baltimore said that he had been interested in a case which possibly might bear on Dr. Elsberg's remarks.

The patient was a school girl who had recently been operated on. Three years ago she had a pain in the left arm which was thought to be due to a toxic neuritis. She complained very little and went on with her school and athletic pursuits, but her hand became slightly atrophic. Three years after the onset she began to notice that she did not use her left leg as well as her right. This became evident about six weeks before the operation.

Her left hand was smaller than the right. She used it awkwardly, but it gave her little trouble. Her left leg showed the evidences of a mild pyramidal tract lesion. There was also a sensory loss of pain and temperature on the right side, involving the second thoracic segment and the whole side below. She had had no pain except that in her left arm three years before.

She was operated on by Dr. Dandy who found inside of the dura a small tumor, opposite the seventh cervical vertebra that hardly indented the spinal cord, but pressed on its left lateral aspect. This intradural tumor was an offshoot of a tumor the size of a lime that was in the body of the seventh cervical vertebra. He believed that the tumor was completely removed.

Dr. Thomas said that while the pressure on the spinal cord seemed to have been recent and slight, the sensory disturbance involved the whole side of the body, up to the level of the segment of the cord pressed on and did not involve the leg only, as might have been expected from Dr. Elsberg's experiences.

Dr. Lewis J. Pollock of Chicago said that it had been noted for some years that in cases of Brown-Séquard's paralysis, occurring as the result of lesions in the lower cervical or dorsal segments, analgesia was frequently absent in the lowermost sacral segments. It had likewise been noted that in patients recovering from this paralysis there was a caudal retreat of the analgesia. From these observations it had been concluded that there was a lamellar distribution of the ascending sensory tracts subserving pain and temperature senses. It had likewise been concluded that the fibers subserving the lowermost segments occupied a position lateral to those of the upper segments.

If this were true, there would be little difficulty in the differentiation of intramedullary from extramedullary lesions. When, in a case in which the lesion was situated in the dorsal or cervical segments, the sacral segments were not analgesic, an intramedullary condition was present.

Dr. Pollock had observed a number of cases of intramedullary lesions and found in them that the sacral segments were free from analgesia. However, in two cases of undoubted extramedullary lesions, one which at operation was found to be a pachymeningitis, these areas were likewise free from loss of pain sense. It seemed that such a condition could be explained more logically on the basis of a wide diffusion of the fibers from the lowermost segments as they ascend than on a lamellar distribution. It might be possible as there does not seem to be a crossing of the pain fibers below the twelfth dorsal segment, that in the lumbar and sacral portions of the cord pain sensation is transmitted through short fibers, as in the cat, in whom a bilateral hemisection of the cord at different levels fails to produce analgesia.

Dr. Charles A. Elsberg said, in answer to Dr. Thomas, that he had operated on several patients in whom tumors had grown through the dura, from the outside, and into the intradural space.

Recently he had witnessed an operation performed by Dr. Taylor, in which a tumor had evidently begun in one of the branches of a spinal nerve or near the spinal ganglion, had grown into the dural sac through the opening in the dura for the nerve root, and had then caused compression symptoms.

Dr. Williams B. Cadwalader of Philadelphia, in closing, said that he was glad his remarks had aroused some interest and thought that the views expressed by Dr. Elsberg regarding the arrangement of the sensory tracts within the spinal cord were most important.

A CASE OF FAMILY PERIODIC PARALYSIS *

M. NEUSTAEDTER, M.D., Ph.D.
NEW YORK

Since comparatively few cases of this disease have been reported I feel justified in recording one recently seen.

REPORT OF A CASE

History.—J. M., a young man, aged 18½ years, a dental student, had a paternal cousin who died of cancer at the age of 30, his maternal grandmother died of diabetes, two maternal cousins were insane, three maternal uncles had periodic family paralysis, of whom one died in an attack at the age of 35, another died of pneumonia at 27, the third is living. He is 29 years old, and has had no attack for three years. The three uncles were not married. In two the attacks began at the age of 16 and in the third at 15. The one who died of pneumonia had the last attack about six months before his death. Otherwise there was nothing of importance in the family history. The parents could not tell whether cases had appeared in previous generations.

He is one of five children. The four are in perfect health. Since early childhood he stammered slightly. Otherwise the history was negative. He had never suffered from constipation.

His first attack was at the age of 14 years, during the evening. It was characterized by marked weakness in all extremities lasting a half an hour. He did not remember any exciting cause. The second seizure at 15, at night, completely paralyzed all extremities. In the third, eight months later, he became paralyzed in the evening and fully recovered in the morning. In the fourth attack, eight months later, he was paralyzed in the morning and did not recover until noontime. In the following one and one-half years he had five attacks at intervals of from three to six months, the last two at intervals of three weeks. All these attacks came on over night and lasted till afternoon. The paralysis was complete, involving all extremities, and the muscles of the trunk and neck. They were ushered in by a feeling of fatigue and numb ache in the extremities and palpitation of the heart, lasting at times a whole day. He would go to bed dizzy, sleep profoundly and in the morning be completely or partially paralyzed in the upper or lower extremities. The paralysis would grow progressively worse until all the musculature above mentioned would become involved. In some of the attacks he had difficulty in deglutition. On recovering, the muscles that became affected last would improve first, the distal ends of the extremities first, then the proximal parts. He invariably remained generally weak for twenty-four hours after a seizure. He vomited a number of times in all attacks, except in the last two. Headache was always present and perspiration was a constant accompaniment. It could not be ascertained whether the temperature was elevated during the attacks.

Present Seizure, February 1. The day before in college he was called on during the quiz hour for the first time. He became very much disturbed and was hardly able to walk to the rostrum. He managed, however, to answer the questions correctly. On returning to his seat he felt the peculiar numb ache of the arms and palpitation, and at once "knew" that an attack was impending. This condition continued throughout the day and evening. His appetite was poor. He did his school work as usual and retired at the usual hour, 11 p. m., slept soundly and awakened at 6 a. m., paralyzed in the lower extremities and trunk. The muscles of the upper extremities soon became involved and then those of the neck. The face, speech and deglutition remained unimpaired. He perspired profusely.

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* Read before the Section on Nervous and Mental Diseases at the Seventy-Second Annual Session of the American Medical Association, Boston, June, 1921.

At 12 m. the temperature by mouth was 100 F.; blood pressure, systolic, 80; diastolic, 60; pulse full and regular, 72; heart normal; respirations 24, fairly deep; the entire body moist.

Reflexes: The pupils were dilated, regular, equal, promptly responding to light and in convergence. The scleral, jaw, pharyngeal, biceps, triceps, radial, abdominal, cremasteric, Achilles and plantar reflexes were absent. The patellars were weak. There was no disturbance of sensation and no response to mechanical stimulation of the muscles. His physician reported that on a previous occasion no response was elicited by faradism.

Motor: The muscles of the neck were fairly strong; eye movements round; musculature of the shoulders flaccid and paralyzed; he was able to flex the forearm, hands and fingers as well as extend them weakly; pronation and supination were executed with some difficulty; respiration was normal, the intercostals and diaphragm were fairly active; the abdominal and dorsal muscles were paralyzed. At times he could flex the right thigh on the abdomen with great difficulty, but not the left. There was marked foot drop on both sides and no movement of feet nor toes. There was flaccid paralysis of the lower extremities. The consistency of his musculature presented no abnormalities. The mucous membranes, abdominal and thoracic viscera were normal. Fundi oculi were normal.

Intellect was intact. His appetite was poor. He passed urine several times; there was no sphincter disturbance.

At 3 p. m. the paralysis was gone; he could rise without difficulty and move all extremities. Motor power was fairly good, but failed after walking about thirty feet. The scleral, jaw, pharyngeal, biceps and radial reflexes were still absent; all others returned. He was still perspiring.

The chemical analysis of his blood by Dr. William C. Thro, showed blood sugar, 130 mg. per 100 c.c.; nonprotein nitrogen, 58.8 mg. per 100 c.c. of blood. The calcium of blood content could not be determined on account of the potassium oxalate, which was added to the blood as an anticoagulant.

Urine: Examination revealed: 1,550 c.c., collected during twenty-four hours; cloudy; yellow, acid; specific gravity, 1.025; indican normal; no albumin, sugar, casts, nor cells; amorphous phosphates and magnesium phosphate crystals present.

Nitrogen Excretion: The total nitrogen excretion was 1.223 per cent.; 18.9 gm. in twenty-four hours; urea nitrogen, 0.965 per cent., 79.0 per cent. of total nitrogen; ammonia nitrogen, 0.04 per cent., 3.2 per cent. of total nitrogen; purin nitrogen, 0.027 per cent., 2.2 per cent. of total nitrogen (+ uric acid); creatinin nitrogen, 0.024 per cent., 1.9 per cent. of total nitrogen; total creatinin nitrogen, 0.372 gm. in twenty-four hours; rest nitrogen, 0.167 per cent., 13.6 per cent. of total nitrogen; creatinin, 1.03 gm. in twenty-four hours.

The patient was put on a Schmidt diet for three days and the feces were examined. The following is the report: Mucus and blood absent; fermentation:

gas + + +, reaction before neutral and after acid. Food residue: muscle fibers very few; small bits of connective tissue, no starch; parasites none, ova none. Bacteria: There were many fine gram-negative rods, very few *Bacilli aerogenes capsulati.* Urobilin normal. The feces show a carbohydrate fermentation type.

February 12, his blood calcium content was 20 mg. in 100 c.c., the highest calcium content ever reported. Oatmeal, milk, eggs and vegetables were eliminated from his diet for a month, and on March 12 his blood was again examined. This time the calcium content varied between 8.6 mg. and 9 mg. in 100 c. c. on repeated examinations. Nonprotein nitrogen now was only 39 mg. per 100 c.c. of blood; about 20 mg. less than on the day of the paralysis; blood sugar 166 mg. per 100 c.c., 36 mg. per 100 c.c. higher than before; creatinin, 1.2 mg. per 100 c.c.

In order to test the possibility of anaphylaxis he was vaccinated with seventy different proteins, consisting of food, epidermal and bacterial proteins, which the Arlington Chemical Company kindly placed at my disposal. All vaccinations proved negative.

March 12, he showed no physical abnormality whatever. There was, however, marked quantitative reduction to electrical stimulation with both currents as compared with another individual of the same age. His systolic blood pressure was 90, diastolic 60, pulse 78. There was a systolic murmur at the apex.

The differential blood count was: polymorphonuclears, 68 per cent.; transitionals, 10 per cent.; lymphocytes, 18 per cent.; large mononuclears, 1 per cent.; eosinophils, 3 per cent.; no basophils and no mast cells. The Wassermann reaction was negative.

April 23, Dr. Selian Neuhof examined the patient's cardiovascular system and reported "No evidence of cardiovascular disease. The abnormal systolic thrill at the apex and the split sound over the mitral area are of functional origin and due to cardiac hyperactivity. These abnormalities disappear almost entirely when the patient is lying down. The systolic blood pressure was at first 140; after a few minutes it fell to 118. This is evidence of heightened vasomotor tone. The diastolic blood pressure was 60. The orthodiascopic tracing shows a slender heart of graceful form, resting slightly on the diaphragm. This is assumed by some to fit in with the complex of vasomotor instability. The electrocardiogram shows normal rhythm. The deviations in the leads are normal in direction and size."

Treatment and Course.—June 4, he had two attacks. He was perfectly well on the previous day, and was sure there was no cause for worry or excitement. At 2 a. m., he woke up perspiring profusely, weak, and with a numb ache in all the muscles, dry throat and dizziness. Although he was not constipated, an enema was given at once, with good results. The weakness, however, progressed, and fifteen minutes after reaching bed he became totally paralyzed from the neck down. An hour later nausea set in, and he vomited three times during the day a fetid, greenish mucus.

At 10 a. m., his physical status was the same as in the last attack, with the exception that the temperature was 97 F. and the systolic blood pressure 140. The involved muscles responded to neither faradism nor galvanism. After a profound sleep, he woke at 3 p. m., fully recovered, but weak.

During the day he took 2 grains of calomel in fractional doses, followed by castor oil. Weak tea with milk was his diet the entire day. At 11 p. m. he vomited again and an hour later he was again completely paralyzed. He slept soundly through the night, arose recovered but weak, and, June 6, was entirely well.

His blood, taken during the attack, showed a calcium content of 4.3 mg. per 100 c. c. The blood taken twenty-four hours after recovery contained 95 per cent. hemoglobin, 6,000 white cells, 40 per cent. polymorphonuclears, 5 per cent. transitionals, 38 per cent. lymphocytes, 10 per cent. large mononuclears, 5 per cent. eosinophils and 2 per cent. mast cells. The urine, 1,000 c. c. collected in the first twenty-four hours, had a specific gravity of 1.025; it contained a trace of albumin and marked increase of indican and a few cylindroids.

DISCUSSION [1]

The etiology of the disease is still obscure, but a psychopathic or neuropathic constitution seems to be the basis. This would be warranted by the familial character of the affection.

To Westphal's theory of a state of toxicity and Goldflam's autointoxication theory Schmidt adds the theory of ischemia, which, while transient in character, produces muscular changes. In such event a temporary lack of nutrition and transient accumulation of toxic materials may partially explain the syndrome. He further contends that an abnormally large amount of epinephrin is secreted in these neurotic persons and this produces the vasoconstriction. It is interesting to note that Orzechowski has produced typical paralysis in a patient suffering from periodic paralysis by injecting epinephrin. The reported glycosuria in some cases and the hyperglycemia in my case would point to a possible suprarenal glycemia.

If the theory of Schmidt is accepted, the specific toxin and autointoxication theories as a terminus ad quem and not a quo would fit into the genesis of the whole picture. The appearance of this syndrome in varying intensity at various ages, especially during and immediately after puberty, in most cases, and intermission during pregnancy may throw some light on this theory. The fact that disturbances of digestion begin and continue with the attack, characterized by nausea, vomiting, constipation, tenesmus, etc., excessive perspiration and also disturbances of heart action would certainly uphold such a theory.

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BIBLIOGRAPHY

Hartwig: Ueber einen Fall intermittierender Paralysis spinalis, Inaug. Diss., Halle, 1874; ref. by Bernhardt: Centralbl. f. d. med. Wissensch., 1875, p. 248.

Couzot: Cas de paralysie périodique, Bull. d'Acad. de méd. de Belgique, 1886, p. 791; Rev. de méd., 1887, p. 190.

Westphal: Ueber einen Fall von periodisch auftretender Laehmung, Berl. klin. Wchnschr., 1885, p. 489.

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[1] Full historical discussion of etiology and pathology of the disease will appear in the volume of transactions of the section on Nervous and Mental Diseases of the American Medical Association for 1921.

Goldflam: Ueber eine eigentümliche Form von periodischer, familiarer wahrscheinlich autointoxicatorischer Lähmung, Wien. med. Presse, 1890, p. 1418; Ztschr. f. klin. Med. 1891, p. 240; Deutsch. Ztschr. f. Nerven., 1895, p. 1; 1897, p. 242.

Taylor: Family Periodic Paralysis, J. Nerv. & Ment. Dis., 1896, p. 637.

Mitchell: Family Periodic Paralysis, Am. J. M. Sc., 1899, p. 513.

Singer and Goodbody: A Case of Family Periodic Paralysis, Brain, 1901, p. 257.

Oddo and Darcourt: Bull. et mém. Soc. de. Biol., Nov. 2, 1901.

Oddo and Audibert: La Paralyse familiale périodique, Arch. gén. de méd., 1902, p. 37.

Crafts and Irvin: A Fifth Case of Family Periodic Paralysis, Am. J. M. Sc., 1900, p. 651.

Mitchell, Flexner and Edsall: Brain, 1902, p. 109.

Cramer: Ref. Neurol. Centralbl., 1909.

Schlesinger: Wien. klin. Wchnschr., 1905, p. 323.

Holtzapple, G. E.: Periodic Paralysis, J. A. M. A. **45**:1224 (Oct. 21) 1905.

Buzzard: Three Cases of Family Periodic Paralysis, Lancet, 1901, p. 1564.

Samuelsohn: Fall von intermittierender Paraplegie, Berl. klin. Wchnschr. **14**:607.

Putnam: A Case of Family Periodic Paralysis, Am. J. M. Sc., 1900.

Oppenheim: Charité Ann., 1891, p. 350.

Schmidt, A. K. E.: Die paroxysmale Lähmung, Monographie 18, Aus dem Gesammtgebiete der Neurologie and Psychiatrie, Berlin, 1919.

Orzechowski: Ref. Neurol. Centralbl., 1909.

Schachnowitsch: Russk. Vrach, 1882, p. 537.

Burr: Periodic Paralysis, Univ. Med. Mag., 1892, p. 836.

Lenoble: Etude sur trois cas des maladie nerveuse familiable, Arch. de neurol., 1901.

Rich: A Unique Form of Motor Paralysis, Med. News, 1894, p. 210.

ABSTRACT OF DISCUSSION

Dr. E. W. Taylor, Boston: Dr. Neustaedter's investigations have added something to neurology. When this subject first attracted attention in Boston we arrived at no conclusion as to the etiology of this extraordinary condition. Dr. Neustaedter has evidently taken a definite step in the right direction, and I have no doubt some sort of definite chemical cause will be found for this paralysis, which stands unique in that during the attack the electrical reactions are entirely lost. It is a loss of muscular reaction, not muscle degeneration. The cases are so rare that they cannot be studied in bulk, but I hope when opportunity offers that Dr. Neustaedter will continue his work along the anaphylactic line of research.

News and Comment

SECOND ANNUAL MEETING OF THE ASSOCIATION FOR RESEARCH IN NERVOUS AND MENTAL DISEASES

The second annual meeting of the Association for Research in Nervous and Mental Diseases will be held in New York during Christmas week. The program contains a symposium of research contributions on the etiology, symptomatology, incidence and pathologic characteristics of multiple sclerosis. Contributors were assigned special topics for investigation early in 1921.

The Commission will be furnished abstracts of papers beforehand, and, as at the last meeting, will put questions to the speakers on the material presented.

The proceedings will be edited and published in book form. The volume on epidemic encephalitis, the topic of investigation last year, is nearly completed and will soon be distributed.

The exact time and place of the December, 1921, meeting will be announced in a subsequent number of this journal.

Abstracts from Current Literature

REPORT OF A NUMBER OF OPERATED VERTEBRAL AND SPINAL CORD TUMORS. G. Mingazzini, Arch. f. Psychiat. **62**:121, 1920.

The writer's cases number six: two vertebral and four spinal tumors. Three of the latter were extramedullary. Two of the patients in the series (Nos. 3 and 4) had extradural echinococcus cysts and both made complete recoveries after surgical treatment.

Case 1 was a caudal perithelioma in a man aged 43, which lay beneath the third and fourth lumbar laminae. It was successfully removed surgically although the patient died some months later from marasmus. The symptoms were initiated by pain in the left lumbar region which extended later to the left nates and finally to the entire right lower extremity. Coincident with the pain in the right lower extremity, flaccid paralysis of the entire left extremity beginning in the left foot ensued, then the right foot became weak, also rectovesical symptoms developed. Examination of active motion showed only a slight extension of the left thigh possible; on the right side flexion of the leg on the thigh was distinctly weak. Riding breeches anesthesia of the sacral roots, hypesthesia of the lower lumbar roots to all kinds of superficial stimuli and loss of inferior tendon reflexes completed the syndrome.

In commenting on this case the writer calls particular attention to the segmental extension of motor paralysis which on the right side is explained as due to a compression of a spinal root contralateral to the tumor, thus differing from a cord lesion proper in which the transition is from monoplegia to complete paraplegia without segmental progression. This case shows that in caudal as well as conus lesions the disturbance of rectal and vesical function may be synchronous.

Case 2 was that of a vertebral sarcoma in a man aged 51 years, involving the lowest dorsal and the first two lumbar vertebrae. It ran a clinical course conveniently divided into three stages: The first stage consisted of lumbar pain and sudden loss of sexual power, separated by a pain-free interval from the second stage of aggravated and intense pain in the hips and the simultaneous loss of inferior tendon reflexes and onset of paraplegia accompanied by atrophy of the right quadriceps. The third stage was marked by retention of urine, pressure pain over the first two lumbar vertebrae and edema of the lumbar spine. The only sensory disturbance present was a band of hypesthesia in the upper and posterior portion of the left thigh for all kinds of superficial stimuli. Active motor function was conserved in the legs and feet, but considerably impaired in the thighs which could neither be completely flexed, abducted or extended. Adduction was possible. Lumbar puncture revealed a fluid of yellow color (xanthochromia) which coagulated spontaneously and also contained an excess of leukocytes. Roentgenograms of the spine were negative. The presence of a positive tuberculin reaction suggested tuberculosis, which, however, was eliminated from the clinical picture. In favor of a vertebral rather than a caudal tumor was the marked rigidity of the trunk and the slight disturbance of the objective sensibility as contrasted with the marked subjective sensibility disturbances.

An attempt at operative relief resulted in determining the character and site of the lesion, which, however, did not involve the dura or interrupt the integrity of the cord or spinal roots. The patient died from cardiac failure at the close of the operation.

The author does not recall in the literature such an edema as he describes, although he quotes Oppenheim and also Bruns as mentioning a widening of neighborhood cutaneous veins in such cases. As an explanation it is suggested that the blocking of the lumbar efferent veins at the intervertebral foraminae may be the causative factor. From the radiographic standpoint this case is quoted in support of the contention of Flatau that the roentgen rays offer little aid in the diagnosis of vertebral tumors. This has been quite opposite to the experience of the reviewer, especially in this region of the spine.

In Case 3, the patient, a woman aged 40, complained first of left lumbar pain radiating into the left hypochondrium, then weakness developed in the left lower extremity rapidly extending to the right lower extremity, finally rendering the patient completely paraplegic. Both patellar reflexes were exaggerated and clonic contractions in the lower extremities, particularly the left lower extremity, were observed. Sensory disturbances reached as high as lumbar 2-4. Three periods of the clinical course were distinguished: neuralgic pain, Brown-Séquard syndrome and paraplegia. The course of the affection was about ten months. Tuberculosis of the spine was first suspected because of a swelling to the left of the spine in the region of the last two ribs. Roentgenograms were, however, negative and it was only by repeated exploratory punctures, the last revealing a gelatinous yellowish fluid containing daughter cysts, that the diagnosis was established. The operation disclosed a cord compression by the cyst which had eroded the arches of the last two dorsal and first lumbar vertebrae but did not perforate the dura.

In Case 4 an echinococcus cyst of the lung had been extirpated eight years before. Pain in the right lower extremity of a sciatica-like character and distribution was the first symptom. Five months after the onset of pain a swelling was noticed at the fifth lumbar vertebra, and this eventually proved to be a recurrence of the echinococcus infection. The neurologic examination at this stage showed absent Achilles' reflexes, hypotonicity of the right calf muscles and a striking weakness in the flexors of the right thigh on the pelvis. Neither atrophies nor objective sensibility disturbances existed. The cyst was found to extend into the last two lumbar vertebrae.

The fifth case reported was that of a woman 45 years old, who five years previously had her uterus removed because of a myoma. An intradural fibrosarcoma compressed the cord at the level of the seventh cervical segment producing a Brown-Séquard syndrome. About a year before coming under medical observation paresthesias were first noticed in the region of the right scapula and somewhat later were felt in the right upper extremity, radiating to the first three fingers. Some eight months after the onset of these paresthesias pain was felt in the right upper extremity, and occasionally in the left upper extremity, in the back of the neck and in the upper part of the sternum. Then paresthesias in the left lower extremity developed and later were felt in the left side of the abdomen. Weakness of the right hand and of the right lower extremity was complained of.

Examination showed a hypotonia of the musculature of the right upper extremity with definite atrophy of the hypothenar eminence and marked loss of strength, especially in the forearm and hand. In the lower extremities drop foot and outward rotation was found on the right side, with considerable weakness in all three segments of this extremity, with especial lack of tone in the right calf muscles. The tendon reflexes on the right were increased over those on the left, and ankle clonus and the Babinski reflex were both present. In the left lower extremity and abdomen marked disturbance of painful and temperature sensation was found. At higher sensory levels the ulnar border of the right hand and forearm presented at times varying degrees of hyperesthesia and hypesthesia; bilaterally in the posterior cervical region a hyperalgesia existed. From the standpoint of the deep sensibility, astereognosis and impaired weight discrimination was found in the right hand, and in the left lower extremity hyperpallanesthesia and parapallanesthesia. The case therefore presented a Brown-Séquard syndrome. The cerebrospinal fluid was not remarkable. The pulse was ordinarily somewhat slow (60) and in the last month of observation a slight elevation of temperature was noted (37.8 C.). The spine was held upright and stiff.

The tumor, 3.5 cm. long, was found beneath the dura by removing the sixth and seventh cervical spines. It was seen to compress the right posterior columns of the cord and the eighth cervical root. The recovery was uneventful, only an atrophy of the right hypothenar eminence remaining.

In the analysis of this case the variations of the subjective disturbances of sensibility illustrated the impossibility of early localization of the tumor by this means. The disturbance of vibration sense due to irritative causes corroborates Frank's postulation that the fibers which transmit vibration ascend homolaterally in the region of the lateral pyramidal tract, and that pallanesthesia is even a more constant symptom in pyramidal tract lesions than the Babinski sign.

The last case of the series, that of a woman aged 29, was similar to the foregoing case in that the cord was compressed by an intradural fibrosarcoma located beneath the fifth, sixth and seventh dorsal spines, producing a Brown-Séquard syndrome. The clinical course could be divided into three stages, the first of radicular pain corresponding to the first and second lumbar roots on the left side. Second Stage: A Brown-Séquard paralysis (objective sensibility disturbance in the right lower extremity and paresis of the opposite extremity). The objective sensory disturbances affected touch, pain and temperature and extended as high as dorsal 9. The motor disturbances in the left leg at this stage were predominately flexor paresis, as flexion of the thigh was only possible to half the normal range. Extension of the leg on the thigh was possible, but not flexion. Limited flexion and extension of the foot was possible, but not adduction or abduction. The left tendon reflexes were exaggerated. Third Stage: Final paralysis of the extensors of the left thigh and complete paralysis below, with bilateral ankle clonus and Babinski sign. Pallanesthesia and bathyanesthesia were present on the left side. Difficulty was experienced in emptying the bladder and in retaining feces.

Although the tumor was entirely removed at operation there remained a spastic paralysis in flexion on the left side or side of the motor paralysis. This was attributed to the long duration of the compression (three and one-half years) as contrasted with the much shorter duration of the previous case.

Schaller, San Francisco.

TUBERCULOTOXIC MENINGITIS. Heinrich Herschmann, Arch. f. Psychiat. u. Nervenkrankh. **62**:3, 1921.

In the clinic for tuberculosis there are many complaints of headache which always causes the staff to be on the lookout for a beginning tuberculous meningitis. In some patients the headaches disappear, and these cases are classed as meningeal irritation or meningismus, without any distinction from tuberculous meningitis and without an investigation into their pathology. Since Quincke, a toxic form of serous meningitis in infections in other parts of the body, such as those in typhus and pneumonia, is well known. The author has written an article concerning meningitis in dysentery.

With regard to the mechanism of the tuberculotoxic effect on the nervous system, there are only meager references in the literature, and on most points there is much controversy. This is discussed in French literature by Laignel-Lavastine and Armand-Delille. The former found in tuberculous meningitis changes in the cells of the cortex which were considered to be independent of the meningovascular processes. The involved cells showed the characteristic signs caused by poisoning. He means by this that the cell changes through the poison of tuberculosis are like those of other poisons of infectious diseases, such as pneumonia. Here Oppenheim gives the anatomic confirmation by stating that the poison can cause direct cerebral symptoms.

The possibility of a direct injury to the meninges by the tuberculosis toxin is up to the present doubtful and a search through the literature revealed the following case of Riebold: A 14-year old boy with bone tuberculosis became ill with meningitic symptoms which disappeared in three days. Four weeks later he developed similar symptoms from which he died very soon. In the discussion the author suggests that at first there was a serous meningitis produced by the tuberculosis toxin and the symptoms quickly cleared up, but this had formed a favorable place for the lodging of the tubercle bacillus. More convincing is the work of Villaret and Tixier who have a more authentic case of tuberculous meningitis in which search was made for the tubercle bacillus, eventually by direct study (stain in sections) and also by animal inoculation, but the germs were not found. They conclude that in this case the process was the result of the toxin. Dr. Herschmann then quotes his own case: A young man, 21 years old, seen Feb. 21, 1919, complained of severe left-sided headache which had been present since November, associated with optical sensations in the form of light zig-zag figures like lightning. Recently he had suffered from loss of appetite, night sweats and a slight cough, but he had had no fever and no brain symptoms. At this time the examination was negative except that the roentgenogram undoubtedly showed intracranial pressure, and there was active tuberculosis of the left apex. Lumbar puncture revealed no pellicle; the globulin was increased; there were 400 cells, of which 90 per cent. were lymphocytes and 10 per cent. polymorphonuclears. No bacteria were found and animal inoculation was negative. Gradually the cell count came down, and the final puncture showed a normal count. He was discharged April 25. In the résumé the author states that it is certain that this patient had a meningitis as shown by the enormous lymphocytosis of the spinal fluid. In contradistinction to the intensely positive findings in the spinal fluid is the poverty of clinical symptoms. Remarkable is the fact that the roentgen ray showed the increase of intracranial pressure three months after the onset. Of great importance is the question of how this peculiar subacute meningeal process had originated. There was no trauma, no disease of the ears and sufficient study showed only tuberculosis of the lungs. The diagnosis of tuberculous meningitis is not verified, but the author quotes Cushing who states that while the lumbar puncture fluid may be sterile, the ventricular fluid may contain the germs, and Foerster's statement that in localized tuberculous meningitis frequently no germs are found in the spinal fluid. The second case quoted is that of a man 51 years old who had suffered for a long time from tuberculosis of the lung. He began to show mental confusion with various delusions, etc. Two children died of tuberculosis. During the last month he complained of headache and was confined to bed for two weeks. On admission he was disoriented. He complained of headache. Examination of the lungs showed a cavernous tuberculosis of both lungs. A few days later he felt pain on pressure over both eyes; the lower extremities were slightly spastic with a Kernig sign on both sides; there was generalized weakness but no paralysis. Otherwise he was normal. Lumbar puncture showed that the spinal fluid was not under pressure and that there was no discoloration. After standing twenty-four hours there was a flocculent precipitate. The Wassermann reaction was negative. There were 43 lymphocytes and 4 polymorphonuclears to each cubic millimeter. The globulin reaction was positive. Bacteriologic study was negative. The patient died of pneumonia a few days later. The microscopic findings in the brain showed that the pia everywhere was considerably thickened—up to from eight to ten times its usual thickness. There were in other parts of the pia portions which were poor in nuclei and dense; there was vessel-free connective tissue which looked like degenerated hyaline tissue. On the inner side there was rich, young edematous connective tissue. The tissue of this zone was filled with blood and greatly infiltrated with cells—in part round, lymphocytic nuclei and in greater part oval or spindle-shaped connective tissue nuclei. The cell forms did not show the characteristic arrangement of tubercles; epithelioid and giant cells were not seen. In commenting on this case, the author states that without the necropsy findings one would consider this a beginning tuberculous meningitis, but the histologic findings show it to be an acute exacerbation of a chronic meningitis.

In conclusion the author states that the findings of Villaret and Tixier, the cases of Riebold and Busse, likewise his own cases, make the existence of a tuberculotoxic meningitis probable.

Winkelman, Philadelphia.

A CONTRIBUTION TO THE PATHOLOGY OF THE HYPOPHYSIS.

Von Monakow, Schweiz. Arch. Neurol. u. Psychiat. **8**:200-207, 1921.

The rôle that the hypophysis plays in producing the condition of Fröhlich's syndrome is to this day not entirely clear. Whether the anterior lobe participates, as Cushing thinks, the pars intermedia, as Biedl thinks, or the neurohypophysis, as B. Fischer thinks, or even whether the hypophysis is at all responsible, is still a mooted question. While we are familiar with the fact that a puncture of several portions of the nervous system will produce metabolic disorders, for instance glycosuria, it must be remembered that we are quite unfamiliar with what portion of the nervous system is injured; whether cells or tracts are irritated, or whether they are destroyed. The relationship of these phenomena to the suprarenals and the liver is, furthermore, unknown.

Von Monakow reports the case of a man, 58 years of age, in whom there was almost complete disappearance of every portion of the hypophysis, with the exception of the posterior lobe, which was practically unchanged. The pathogenesis of this lesion was not clear. It is possible that it was secondary to a slight internal hydrocephalus, although the patient had never given any clinical evidence of this disorder, and the necropsy findings were very slight. There was no evidence of tumor anywhere.

The clinical course was as follows: The patient was first seen four years previously, at which time he was suffering with an acute articular infection of unknown etiology. It was noted at the time that his skin was a little dry and atrophic; the testicles were slightly diminished in size; there was a slightly active apical tuberculosis, slight weakness of the facial muscles and increase of the right patellar reflex. Four years later the patient showed marked changes. There was complete disappearance of all hair except that on the head; the skin was dry, thin and alabaster in color; the testicles were extremely atrophic and on section showed not only disappearance of the seminiferous tubules and absence of all spermatozoa, but also complete obliteration of the interstitial cells of Leydig; the penis and prostate were practically unchanged; there was a marked disproportion between the amount of fat and the actively tuberculous process, which was responsible for the fatal termination through peritoneal involvement. Although there was a daily fever and the patient took comparatively little food, he lost scarcely any in body weight, and the panniculus adiposus was everywhere well developed and presented an eunuchoid distribution. Examination of the urine was practically negative; blood chemistry normal; 3,200,000 red cells, hemoglobin 55 per cent.; blood pressure normal; the Wassermann test was negative. It may be added that the fundus examination and roentgenogram of the sella were normal. The administration of 150 gm. of glucose and the injection of 1 mg. of epinephrin caused no glycosuria. Blood pressure, also, was not altered. The administration of thyroid extract did not change the pulse rate, blood pressure or the amount of urine. Pituitary extract had not the slightest effect.

There were two factors of great interest. The first was the peculiar psychic attitude, which was not dependent on any structural nervous lesion of an ordinary character. The patient was markedly apathetic; sexual desire, which had been normal, there having been five healthy children, had diminished for the past six years and was now completely absent. He became indifferent and somewhat stupid; he reacted little to his environment and to his own situation; he never complained; he had no appetite and never called for fluids. His apathy was so marked that he made no attempts to brush away flies, which at times completely covered his face. Answers to questions, however, were always clear and relevant. There was marked physical weakness, which could not be explained on the basis of tuberculosis or the general condition of the muscles.

The second point of interest was the urine output. The hypophysis is looked on by some as giving rise to a hormone that influences the kidney in an inhibitory manner. The disappearance of this hormone, which is supposed to rise from the pars intermedia, should produce a diabetes insipidus. While the pars intermedia was completely absent, he at no time had polyuria; on the other hand he was oliguric, excreting about from 400 to 500 c.c. of a specific gravity of 1.016 to 1.018 daily, a phenomenon that ran parallel to the general slowing down of all vegetative functions. Only shortly before death was there an albuminuria and cylinders and fat granules were found in the urine. At necropsy marked degenerative changes were noted in the kidneys, indicating the presence of a nephrosis. It would appear, from experimental work as well, that there is a relation not only between the hypophysis and other endocrine glands, but also between hypophysis and the kidney, although this point of view must still be considered purely hypothetical.

Woltman, Rochester, Minn.

COMPRESSION SYNDROME IN CEREBROSPINAL FLUID WITH SPECIAL REFERENCE TO THE QUECKENSTEDT SYMPTOM.

Karl Crosz and Martin Pappenheim, Deutsch. Ztschr. f. Nervenh. **67:** 1921.

A voluminous literature has developed on the so-called compression syndrome in the cerebrospinal fluid since Nonne, in 1908, first drew attention to its diagnostic importance. Albumin has been noted in great quantities. In a case of von Reichmann's it reached 4.6 per cent. At times there is spontaneous coagulation and in some instances xanthochromia. The syndrome is found in extradural, intradural and even in intramedullary tumors, in the latter cases being probably brought about through a collateral edema. It has also been found in cases showing tubercles, spondylitis, cysts, echinococcus (Queckenstedt), cysticercus (Goldstein), meningitis serosa and serofibrinosa circumscripta, funicular myelitis and in some cases in which diffuse disseminated necrotic changes have occurred, as in different forms of subacute chronic meningitis. The syndrome, namely, high albumin content with normal or slightly increased cell count and at times spontaneous coagulation and xanthochromia, is diagnostic of some obstructive process in the subdural space.

Froin and Ledoux report a case of gumma with meningitic adhesions (necropsy), the fluid of which showed xanthochromia and spontaneous coagulation.

Queckenstedt described a symptom present in cases of obstruction, namely, that pressure on the veins of the neck either failed altogether to produce a rise in the fluid pressure after lumbar puncture or at any rate such rise was delayed and insignificant, in marked contrast to the rapid rise seen in normal cases. He reports five such cases. Later von Redlich described a case of tumor with a positive von Queckenstedt symptom, and von Pappenheim reported it in a case of tuberculous meningitis. Others have also reported this symptom.

The authors' series consists of 14 cases showing symptoms of obstruction, 8 of which were diagnosed as tumors of the dorsal portion of the spinal cord, 1 a tumor of the lumbar cord demonstrated at operation, 1 a cervical tumor (necropsy), and 4 cases of vertebral caries—2 cervical and 2 thoracic—3 of which were confirmed at necropsy. In 2 of the tumor cases there was a slight cell increase of 12 and 13 cells per cubic millimeter. The spinal fluid pressure was measured and Queckenstedt's test was performed in 9 cases, namely, in 4 cord tumors of the dorsal region (3 verified), 1 cervical cord tumor, and in all 4 cases of vertebral caries. The Queckenstedt symptom in the 4 cases of caries and in the 3 verified tumors was positive. In 1 case of a tumor of the eleventh thoracic vertebra in which the albumin content of the fluid was normal, it was the only positive symptom. In another case with a Brown-Séquard syndrome (D 6 — D 7) it was only faintly positive. In a case of paraparesis a positive Queckenstedt symptom became negative after a small amount of fluid had been withdrawn.

The fluid pressure in a few cases was high. In one case of caries of the dorsal region it registered 220 mm. of water and in two cases of tumor 240 mm. and 260 mm. respectively. In the others it was normal. In the foregoing case of caries the pressure dropped rapidly from 220 mm. to 110 mm. after 5 c.c. of fluid had been withdrawn. There is no explanation given for the increased pressure in these cases of obstruction; however it was noted that in two of the three cases with increased pressure the obstruction was not complete.

An increase in globulin and total albumin as determined by the Ross-Ione and the Nonne-Appelt and Pandy methods was noted in twelve cases. In general, the total albumin was not over 2 per cent., but in one case of cervical caries it amounted to 4 per cent. In the tumor of the lumbar cord there was a total albumin content of 1 per cent. with spontaneous coagulation, and this was the only case in the series which showed xanthochromia. In the literature xanthochromia has been reported with relatively greater frequency, and varied explanations have been offered concerning its nature and cause. It is generally conceded, however, to be a hemoglobin derivative gaining access to the fluid either through actual hemorrhage or by passage through the undamaged vessel walls.

The colloidal gold reaction in the patient with xanthochromia and in the patient with caries with great increase in albumin content showed a meningitic curve.

In a case of cervical caries fluid taken during operation from above the obstruction proved to have a normal albumin content which was in marked contrast to the fluid from below the lesion. Others have made similar observations.

The fact that cases of brain tumor almost regularly show similar spinal fluid changes to the ones described render these tests of no differential value. The authors report seven of a series of eight brain tumors showing a moderate increase in albumin of from 0.5 to 1.5 per cent.

The French lay emphasis on a syndrome consisting of increased albumin content with spontaneous coagulation and xanthochromia, first described in 1903 by Froin. At first, hemorrhagic processes were thought to be the cause, later it was deemed to be due to low pressure in the sac which caused changes in the vessel walls and allowed exudation. By some it was considered to be a result of fluid stasis. Baruch assumes that the tumor itself secretes an albuminous fluid.

Regarding the nature of the compression the French give three possibilities: pressure from without, fibrous, meningomedullary adhesions, and acute inflammatory conditions.

In conclusion, the authors add five cases as further proof that the compression syndrome when found in inflammatory conditions may not be due solely to this pathologic process but also to an actual obstruction as demonstrated by a positive von Queckenstedt symptom. Case 1: Tuberculous meningitis. The pressure was 220 mm. which dropped to 70 mm. after 6 c.c. of fluid had been drained off; positive Queckenstedt symptom, xanthochromia, 70 cells per cubic millimeter, 40 per cent. of which were polymorphonuclears, strong phase 1. The next day the pressure was 180 mm., which dropped to 0 after 5 c.c. of fluid had been withdrawn. There was marked xanthochromia, 60 cells per cubic millimeter, total albumin almost 2 per cent., with spontaneous coagulation. Necropsy showed extensive exudative meningitis. Case 2: Epidemic meningitis showing light colored fluid, pressure 100 mm., positive Queckenstedt symptom. Following the removal of 2.5 c.c. of fluid the pressure dropped to 60 mm. There were 700 cells, 60 per cent. of which were polymorphonuclears. Strong phase 1, albumin content about 6 per cent. Case 3: Purulent meningitis (following injury); turbid fluid, pressure 220 mm., which dropped to 170 following the withdrawal of 3.5 c.c. of fluid, positive Queckenstedt symptom, several thousand cells chiefly of the polymorphonuclear variety; albumin about 5.25 per cent. Case 4: Positive Queckenstedt symptom in a case showing localizing symptoms in the lower thoracic region. The patient admitted syphilitic infection nine years previously. At present the blood Wassermann reaction was negative, but the spinal fluid was + + +. The fluid pressure was 60 mm. which dropped to 0 on the withdrawal of 3 c.c. of fluid; globulin and albumin increase 3 per cent.; 185 cells per cubic millimeter; no xanthochromia. The patient improved under treatment. Case 5: First seen by von Muller who noted at operation inflammatory changes with adhesions of the cord membranes. The authors later performed a spinal puncture on the same patient and noted a normal fluid pressure, a markedly positive Queckenstedt symptom, 16 cells per cubic millimeter, and slight increase in globulin and albumin.

Nagel, San Francisco.

BASEDOW'S SYNDROME WITH TROPHEDEMA. C. J. Parhon and A. Stocker, Rev. neurol. **27**: 1020 (Oct.) 1920.

The association of hyperthyroidism with trophedema impresses the authors as of sufficient significance to justify report, particularly so since the mention of twenty-five cases in the literature to date indicates the possibility of more than merely coincidental association between the two conditions.

Their case was that of a girl 21 years of age. The family history was negative, with the exception of alcoholism in one grandfather, tuberculosis in one brother and the existence of a psychosis (type unspecified) in a paternal step-brother. The personal history was negative until the beginning of the present trouble except that during childhood she had diphtheria, whooping cough, measles and malaria: The present trouble dated back to a severe affective shock sustained two years previously as a result of a "Bolshevik" raid in which a member of the patient's party was murdered, the patient, herself barely escaping with her life. This strain was definitely aggravated soon after by the necessity of testifying at the trial of the guilty parties. As a result, the patient was profoundly prostrated and in addition to general physical and nervous weakness there were noted crying spells, irritability, cardiac distress, vomiting and insomnia. After some time the thyroid was observed to enlarge, particularly on the right, and there was marked protrusion of the eyeballs. The patient also complained of headaches and distressing pulsation in the right arm and right side of the face. There was also thinning and lightening of the hair and eyebrows. Some time later the patient noted a swelling about the ankles, which ultimately involved both legs. There was cessation of menses for two years, from the time of the accident until the patient entered the authors' service.

On examination the patient complained of marked weakness and ready fatigability and showed marked exophthalmos, Dalrymple sign, marked thyroid hypertrophy, particularly of the right lobe and the isthmus, thinning and lightening in color of the hair and eyebrows, somewhat accelerated respiration (22) accompanied by a feeling of thoracic oppression and occasional dyspnea. The heart rate was 120, and there was complaint of constant distressing palpitation. The extended fingers showed marked tremor. The legs showed increased circumference to a level just above the knees and presented a somewhat edematous appearance, although there was no evidence of pitting following pressure nor any indication of temperature or color change. The condition seemed essentially a uniform subcutaneous or areolar fatty infiltration; and suggested itself to the author as typically trophedema. As a result of this condition the feet were held in a slightly extended position and flexion movements seemed somewhat difficult. The patient complained of ready fatigue on movement and was able to change from one position to another only after great effort. She complained of a feeling of heaviness in the legs and pain in the tibio-tarsal articulations, which was definitely accentuated on movement. There was no change in reflex response except that the knee jerks seemed somewhat diminished. Differential blood count showed only 58.5 per cent. polymorphonuclears of which 5.2 per cent. were eosinophils. The mononuclear cells were mainly of the large type.

In discussing this association of hyperthyroidism and trophedema, on the basis of their own case and those described in the literature, Parhon and Stocker feel that the latter, mainly because of its segmental nature, was induced essentially as a result of vegetative nervous system disturbance, probably intramedullary in locus, which was determined primarily by the existing endocrine imbalance or dystrophy.

Raphael, Ann Arbor, Mich.

COMPRESSION SYNDROME OF THE CEREBROSPINAL FLUID IN TUBERCULOUS SPONDYLITIS. W. Ravens, Deutsch. Ztschr. f. Nervenh. **68**:69, 1921.

The author begins by stating that he has previously been able to report a few cases of tuberculous spondylitis in which the spinal fluid showed a positive phase, one without cell increase, with at times xanthochromia. Among others, Henkel, Schwartz, Bremer, Queckenstedt and Zaloziecki have reported similar findings. Sicard and Foix report nine cases of dorsolumbar pachymeningitis following Pott's disease, in which the spinal fluids were of light straw color, with increased albumin content but no increase in cells. Schnitzler found a positive Phase 1 in a suspected case of cord tumor which proved to be vertebral caries at operation. This led him to make a fluid examination in seven other cases of Pott's disease. Three of these presenting symptoms of cord pressure showed a typical so-called compression fluid, namely, high albumin content with normal cell count, while four uncomplicated cases had normal fluids.

The results of spinal puncture in forty-one personally observed cases of Pott's disease are reported. Twenty-seven patients were male and fourteen female; twenty-five were between the ages of 3 and 10 years; eleven from 11 to 20 years, and five from 20 to 42 years. The disease affected the cervical region in two, the thoracic in twenty-seven, and the lumbar region in twelve instances. The danger of puncture was not considered great in these cases and no ill results are reported.

Twenty-three of the patients showed no cord symptoms and had normal fluids, fifteen had distinct changes in the fluid and three had marked changes. All of the latter eighteen cases had more or less marked symptoms of cord pressure varying from a complete spastic paraplegia with bladder and rectal symptoms to minor local sensory disturbances. In all these eighteen cases the spinal fluid was clear, colorless, with normal or increased pressure. There was a definitely positive Phase 1 in all and it was marked in three cases. There was no cell increase in any of the forty-one cases.

The author concludes that fluid changes in certain cases of spondylitis do not differ qualitatively or quantitatively from those found in tumors which cause obstruction to the flow of the fluid. This fact is considered of more theoretic than practical interest.

The main points of the article are thus summarized:

1. In tuberculous spondylitis the same fluid changes may occur as in tumors compressing the cord.
2. These changes occur in those cases in which there has been pressure on the cord.
3. This fact will not greatly increase the difficulty of differential diagnosis.
4. The degree of increase in albumin runs parallel with the severity of the nervous symptoms.
5. With improvement in clinical symptoms the total albumin content diminishes.

Nagel, San Francisco.

THE STATIC OR POSTURE SYSTEM AND ITS RELATION TO POSTURAL HYPERTONIC STATES OF THE SKELETAL MUSCLES. J. Ramsay Hunt, Neurol. Bull. 3: No. 6 (June) 1921.

In discussing a case of decerebrate rigidity, Hunt reviews his conception of the static and kinetic systems, the various disturbances of motility and posture and considers the different forms of hypertonicity. The following is a brief summary of his views, which he has already stated on previous occasions.

The kinetic system concerns itself with active movement, the static with passive movement or posture. Both active motility and posture may be divided into reflex, automatic-associated and isolated-synergic or dissociated. These types of movement may be correlated anatomically with the reflex or segmental nervous system, with the paleo-encephalic (corpus striatum-optic thalamus) and neo-encephalic (cortical) mechanisms for the kinetic system and the paleocerebellum (vermis, its nuclei and pathways) and neocerebellum (celebellar hemispheres) for the paleostatic and neostatic systems. The paleokinetic and neokinetic systems are subserved, respectively, by the pallidal and pyramidal systems. The afferent pathways for the static system are by way of the corticopontocerebellar and spinocerebellar pathways while the efferent pathways of the neostatic and paleostatic systems, respectively, are by way of the superior peduncle to the nucleus parvicellulatus and nucleus magnocellulatus of the nucleus ruber. Hunt postulates the existence of distinct efferent fibers in the peripheral nerves and separate endings in the muscle spindles, each subserving the static and kinetic systems, and he points to the sarcoplasm and anisotropic disks and the demonstration of nonmedulated fibers in the peripheral nerves to prove his conception of the double motor innervation, thus showing the existence of two systems all through the nervous system even to the final motor end plates. Without going into too many critical details, it may be pointed out parenthetically that the demonstration of nonmedullated nerve fibers and distinct end plates, which speak for double motor innervation, has been shown to refer not to the static or postural mechanism which Hunt conceives of but to the sympathetic nervous system. (See the recent article by J. T. Wilson on the "Double Innervation of Striated Muscle," *Brain* **44**: Pt. 2, 1921.)

Based on the foregoing anatomic-physiologic considerations, Hunt next considers the various postural hypertonicities: first, the cortical or pyramidal, involving mainly the isolated-synergic movements; which explains why the spastic states are so sharply localized in the distal portions of the extremities; second, the striatal or pallidal types of hypertonus with waxy rigidity, loss of automatic-associated movements and lack of the elements of spasticity, such as clonus, hyperreflexia, etc.; third, combined pallidopyramidal types of paralysis and spasticity-rigidity; fourth, midbrain or paleocerebellar type of postural hypertonus (decerebrate rigidity as described by Sherrington and Wilson) which involves the severance of the pyramidal tracts (neokinetic) the pallidal system (paleokinetic) and the cortico-ponto-cerebellar mechanism (neostatic), leaving intact the peleo-cerebello-rubro-spinal system (paleostatic). "Decerebrate rigidity may, therefore, be regarded as the postural expression of paleostatic activity." Finally Hunt considers the spinal type of postural hypertonus which expresses itself on the one hand through paraplegia in flexion, due to a transverse cord lesion and involvement of all long tracts, and paraplegia in extension following involvement only of the pyramidal tracts.

WECHSLER, New York.

METHODS TO SECURE END-TO-END SUTURE OF PERIPHERAL NERVES. HOWARD C. NAFFZIGER, Surg., Gynec. & Obst., March, 1921.

Numerous methods of filling in gaps occasioned by nerve division have been recommended by surgeons and, in large part, have resulted in connecting the severed nerve ends by a material not penetrable by the neurons. The experimental works of Huber, Nageotte and others in the use of free nerve grafts have pointed the way to a more rational treatment. With improvement in operative procedures, the necessity for grafts has become considerably decreased, while end-to-end suture has often become more possible.

More than one hundred patients with peripheral nerve injury have come to operation within the past eighteen months from the United States Army Letterman General Hospital, the neurologic service of the University of California Hospital, and from other sources largely industrial. Autonerve grafts, homografts, or heterografts should be reserved for cases in which all methods of securing direct suture have been exhausted. Apparently it is not an uncommon procedure for surgeons to resect bones of the extremities to secure shortening in order that they may obtain end-to-end suture. Conditions justifying this measure must be exceedingly rare.

The most useful methods in obtaining apposition of the divided nerve ends are: (1) free mobilization of the proximal and distal portions of the nerve, (2) transposition of the nerve to a shorter route than the normal one, (3) favorable posture of the extremity to shorten the distance to be overcome, and (4) gradual lengthening of the nerve by a two-stage operation.

Free mobilization of the nerve requires above all long incisions. Small incisions and forcible stretching of the nerve produce unnecessary trauma and does not secure the greatest lengthening.

Transposition of the nerve to a shorter route than the normal can be employed in the ulnar nerve and in the musculospiral, the ulnar nerve being elevated and placed internal to the condyle and the musculospiral placed anterior to the humerus on the brachialis anticus.

Great stress is placed on choosing a favorable posture to give the greatest mobility of nerve eminence in order to permit end-to-end suture, rather than using nerve graft. The author resorts to gradual lengthening of the nerve by a two-stage operation, in extreme cases suturing the severed ends before the neuromas are excised, placing the extremity into a cast or frame, and permitting gradual stretching for a period of two months. End-to-end anastomosis should follow the removal of the neuromas and without rotation of the nerve axons. The suture line should be surrounded with foreign material, but, if convenient, the suture line should be brought up against uninjured muscle or between muscle planes.

If the arm has been held in flexion or adduction in order to permit an end-to-end anastomosis, it should not be straightened for a period of from five to six weeks and then very gradually in order to permit the extremity to lie in a half cast. Daily massage and gentle passive motion are valuable.

The value of nerve graft is recognized when end-to-end suture cannot be obtained. Such cases, however, are relatively rare and nerve graft should be resorted to only after all attempts at end-to-end suture have been unsuccessful. For the ulnar nerve, mobilization alone permits of suture after a 3 cm. gap; after mobilization and transposition 4.5 cm. Adduction of the arm to the side permits a slight additional gain; with mobilization, transposition, adduction and complete flexion of elbow and wrist, 10 cm. For the musculospiral nerve, mobilization alone permits 2 cm.; with mobilization, elbow flexion and adduction, slightly more, usually about 8.5 cm.; mobilization, transposition, elbow flexion and adduction, 10 cm.

In conclusion, the author states that end-to-end suture is the method of choice and with few exceptions can be employed. This is particularly true of the main nerve trunks. Familiarity with these methods in peripheral nerve surgery gives an increasingly high proportion of end-to-end sutures.

Adson, Rochester, Minn.

STUDIES IN EXHAUSTION: AN EXPERIMENTAL RESEARCH.

G. W. Crile, Arch. Surg. **2**:197 (March) 1921.

Dr. Crile had in mind in this research problem, the identification of the organs and tissues affected by various causes of exhaustion, and whether or not all known causes of exhaustion produce identical end-effects in the essential organs.

He states that stimulants are valueless in exhaustion, that restoration time is much the same for all causes and that sleep is the essential for restoration. The primary cause of exhaustion states could not be ascertained.

In the study of insomnia tests were carried out on normal, healthy rabbits. Early and late histologic, biochemical, physical and chemical changes were observed after subjecting the animals to varying degrees of insomnia. The results of natural sleep, artificial, diurnal and seasonal sleep are compared. The animals were kept continuously awake for from ninety-six to one hundred and eighteen hours, without other disturbances and with plenty of food and water at all times.

In the histologic studies the tissues found to be affected were the central nervous system, the liver and suprarenals, all others showing no change. In the nervous system the cortex was more severely affected than other areas, but since the Purkinje cells of the cerebellum showed changes identical with those of the cortex the differential counts were made from these. In the liver the cells near the periphery of the lobules showed most alteration, while in the suprarenals the cells of the cortex alone were affected.

In the differential counts there were recognized active cells, fatigue cells and exhausted cells. It was found that there was early a considerable increase of active cells above the normal (64.8 per cent.). This is termed a period of overreaction or hyperchromatism. The exhausted cells began to increase again after the period of maximum reaction. Animals in one series that were not killed died soon after the period of insomnia was over. No cause for death could be found and it was concluded that the exhaustion had been carried to a nonsurvivable limit. It is estimated that survival either in man or animal cannot occur after a loss of 15 per cent. of brain cells.

Observations were made during the periods of insomnia and at varying periods afterward. The cell changes consisted mainly of swelling and enlargement of the cells, diminution and disappearance of granules (except in the early hyperchromatic stage), dislocation of nuclei, rupture and disintegration of cells. In regard to the possibilities of restoration, one seance of sleep was found to restore all cells except those disintegrated. In those animals dying from the insomnia, 15 per cent. of the brain cells were fully exhausted. In the cases in which artificial sleep was produced (nitrous oxid-oxygen anesthesia) 90 per cent. of the cells became hyperchromatic. From the latter it was concluded that sleep repairs all cells except those in which the nucleus has disintegrated, and that artificial sleep is markedly beneficial.

From the physiochemical point of view hydrogen-ion concentration determinations were made, it being known that intravenous injections of sodium phosphate or hydrochloric acid produced lesions in the brain, liver and suprarenals identical with those produced by exhaustion. The hydrogen-ion concentration was determined in six rabbits before and after insomnia. The conclusions arrived at were that prolonged insomnia alone produces relatively slight or no alteration.

Electrical conductivity experiments led to the conclusion that prolonged insomnia (ninety-six hours) decreased the conductivity of the central nervous system. After a period of rest (six hours), the conductivity of the cerebrum and cerebellum were decreased still more than at the close of the insomnia period, but the spinal cord had begun to return toward the normal. After a prolonged rest (from nine days to two weeks) the cerebral conductivity was increased above the normal, while that of the cerebellum and spinal cord approached normal.

The body temperature in exhaustion in 42 cases was found to rise an average of 1.5 F. in 24 cases; to fall 0.9 F. in 14 cases; while in 6 there was no change. The respiratory rate showed an average increase of 47 per minute in 6 cases, a decrease of 46 in 31 cases and no alteration in 5 cases. The weight in 23 cases showed an average gain of 107 gm.; a loss of 107 gm. in 18 cases, and no change in 1 case. Urinalysis in 17 cases was negative for pathologic constituents.

The article is illustrated by photomicrographs of the cellular changes, and there are tables of each experiment which are worthy of careful study. In the light of the results obtained an interesting speculation arises concerning the condition of the essential organs in those cases of maniacal excitement in the manic-depressive psychoses, in which patients go for days and even weeks without sleep or rest and are subjected to the most extreme grades of emotional imbalance (which the author was careful to avoid in his experiments), which apparently so completely controls their activities. Neuropathologists are accustomed to the pictures of marked cellular changes in toxic states and terminal conditions. Can death be due to the toxicity of the disease or does exhaustion beyond a survivable limit bring about the fatal termination? In the treatment of all acute disturbances, physical as well as mental, are not sleep and rest, however produced, the greatest therapeutic agents at our command? The author's experiment has certainly opened up a field of inquiry which is rife with possibilities.

Patten, Philadelphia.

A CASE OF PSEUDOTETANUS (ESCHERICH). T. Reh, Rev. neurol. **27:**1017 (Oct.) 1920.

In presenting his case, the author remarks on the apparent clinical infrequency of this condition, only twenty cases having been described thus far in the literature.

Reh's patient, a girl of 3, was brought to his clinic three days after the onset of a series of severe clonic seizures, at first occurring every fifteen or twenty minutes, but by the time of presentation having become somewhat less severe and definitely less frequent. The condition was reported to have been ushered in by cervical rigidity, which gradually became generalized. The family and personal history was entirely negative with the exception of a report of a small cutaneous laceration (lesion still in evidence) received some two weeks previously as a result of a scratch from a ground bramble.

Examination showed in addition to occasional and rather mild clonic seizures, a marked generalized, plank-like rigidity affecting the entire body except that the ocular muscles were practically uninvolved and those of the arms only slightly so. This condition was apparently unaccompanied by pain or any indication of acute subjective distress. The eyelids were noted to be half closed and the eyebrows drawn, with marked wrinkling and creasing of the forehead and cheeks. Trismus was definitely present. Physical and neurologic examinations were otherwise negative, and there was no indication at this time, or at any other during the later course of the illness, of the Weiss, Chvostek or Trousseau signs or evidence of changed electrical reaction, or report of fever. The spinal fluid was negative, and it was impossible to isolate the tetanus bacillus from the skin lesion. Shortly after examination 20 c.c. of tetanus antitoxin were administered.

The clinical course was one of gradual and uneventful improvement, the patient having completely recovered by the twenty-eighth day after admission to Reh's service. Nosologically the author concludes that, although the clinical duality of tetanus and pseudotetanus seems fairly well established, the etiologic status of the latter is still definitely obscure, and he recommends that this condition be regarded rather as a clinical syndrome dependent possibly on a number of etiologic factors than as a truly specific disease entity.

Raphael, Ann Arbor, Mich.

SYPHILITIC BACKACHE: A SYMPTOM OF SYPHILIS OF THE SPINAL CORD, LUMBAR MUSCLES AND VERTEBRAE, WITH REPORT OF CASES. J. V. Klauder, Arch. Dermat. & Syph. **3:**761 (June) 1921.

Klauder discusses backache from the standpoint of neurosyphilis, syphilis of the muscles, vertebrae and synovial involvement of the joints. Most of the syphilitic backaches arise from syphilitic involvement of the spinal cord. A meningeal inflammation causes irritation of the posterior roots or the inflammation may extend to the root itself causing pain. Vascular disease, localized gummatous processes or syphilitic periostitis by extension may cause pain. Meningeal irritation and inflammation is more extensive and intense in the posterior columns and surfaces of the cord, the dorsal region having least resistance suffering most. Meningeal inflammation may exist from the exanthematous period of syphilis and last many years. Headaches and backaches of secondary syphilis are frequently meningeal in origin. The meningeal syndrome is: pain, paresthesias, hyperesthesia and increased tendon reflexes. The patellar tendon reflexes are often variable, due probably to the swelling around the roots from the acute syphilitic process. The early opinion that syphilitic pain was worse at night was not borne out in the author's experience. The patients themselves complain chiefly of backache, fatigability and stiffness. Objectively there are found spinal rigidity, localized or diffuse tenderness on percussion over the spinous processes and painful movement. Extension of the syphilitic process causes the familiar picture of meningomyelitis. Antisyphilitic therapy produces a response proportional to the duration and the stage of the disease, and spinal fluid examinations are frequently positive when the blood Wassermann reaction is negative.

Lumbar muscle involvement occurs in the secondary stage of syphilis, either meningeal in origin or as a spirochetal toxemia with fever, headache and malaise (simulating typhoid). The pathologic changes are: interstitial myositis —attended by swelling and pain, gummatous formations—localized or generalized and accompanied by pain, infiltration and swelling and perhaps ulcer formation.

Vertebral involvement is rare in syphilis, but when it occurs it is usually in the cervical region. It is seen as a periostitis or osteitis. The predominant symptom is pain—acute and lancinating; there are also tenderness, rigidity and obliteration of the normal curve. Deformities occur. Differentiation from tuberculosis is the main concern in diagnosis. Frequently, however, syphilitic infection serves to light up a quiescent tuberculous process.

Syphilitic synovitis is the commonest lesion of spinal syphilis. It occurs early in the disease, affects three or four vertebrae and has for symptomatology: deformity, localized stiffness and hypotonicity of the uninvolved parts. Roentgen-ray evidence is usually wanting.

Case reports are given illustrative of each type of syphilitic involvement and photographs accompany the text.

Patten, Philadelphia.

THE FINGER-FLEXION SIGN IN HEMIPLEGIA. T. Urlatzeanu and D. Noica, Rev. neurol. **27**: 1012 (Oct.) 1920.

The authors direct attention to a feature especially noted by them in recent hemiplegias, dependent on cerebral embolus, thrombosis or hemorrhage, which they believe to be of definite diagnostic significance. Thus it will often be observed, in such patients, that when in the supine position, with the hand of the affected side resting on the bed in slight pronation, it will be seen to be supported on its cubital border with the thumb approximated to the index finger with consequent prominence of the thenar eminence and wrinkling of the skin over the palmothenar area. The terminal phalanx of the thumb is gently flexed in contradistinction to the normal position, that of extension. The other fingers, particularly the last two, are approximated to one another and are held gently flexed. The forearm at the same time is generally found to rest in a position of semipronation. Later in the condition, if recovery does not take place, permanent contracture in this position frequently occurs. This flexion attitude tends to reinstate itself automatically if altered by the examiner. It has been observed that, if the arm is set at an angle of 90 degrees from the trunk, and allowed to hang over the side of the bed, although the forearm and hand are now in supination, the fingers still retain their former flexion position. Urlatzeanu and Noica have been unable to determine any analogous condition as occurring in normal persons, although a similar state, but bilateral, has been noted by them in patients in a state of anesthesia or in coma, either toxic or infectious. The exact mechanism underlying this posture sign is somewhat obscure, although the authors suggest relation, in this connection, to lesion in the opposite cerebral hemisphere.

Raphael, Ann Arbor, Mich.

AN EXTENSIVE SPINAL ARACHNOID FIBROBLASTOMA. Hubert S. Howe, Neurol. Bull. **3:** No. 6 (June) 1921.

Howe reports an unusual type of cord tumor which involved practically the whole cord from the filum terminale up to the lower cervical segments. The condition lasted twelve years from the onset of the first symptom to the date of the patient's death. The first symptoms were those of a tumor of the cord, which though apparently incompletely removed (seventh cervical to the fourth dorsal) resulted in complete clinical recovery for nearly five years. A second operation, eight years after the first, resulted in fair recovery, and a third, three years later, ended in the patient's death. At necropsy the cord was found "imbedded in an irregular tumor mass which was most abundantly present in the sacral region and about the filum terminale, but extended upward as high as the upper dorsal region." Tumor tissue was found also near the fourth cervical segment, although it gave no signs of compression. The unusual features of the case were the immense dimensions of the tumor and almost complete recovery following incomplete removal on two occasions. Another unusual feature, which is difficult to correlate with the involvement of the cord, was the presence of choked disk in the absence of signs of brain involvement and negative necropsy findings on gross examination. An undetected metastatic focus in the brain may possibly be conjectured as the cause of the choked disk, but the findings recorded hardly warrant such a conclusion.

Wechsler, New York.

HISTOLOGIC EXAMINATION OF THE NEURAXIS IN A CASE OF SYDENHAM'S CHOREA. P. Marie and C. Tretiakoff, Rev. Neurol. **27:**428 (May) 1920.

Marie and Tretiakoff present the case of a girl, aged 10, who died ten days after the onset of what appeared clinically as typically Sydenham's chorea, the pathologic findings in which agreed very well, on the whole, with those determined in similar cases previously reported.

Histopathologic study revealed a somewhat diffuse and irregularly distributed inflammatory reaction, primarily perivascular, but also showing definite parenchymatous invasion (particularly the gray matter) and affecting the entire neuraxis (except the bulb and cerebellum), focusing maximally, however, on the basal ganglions—in short, a picture essentially that of a polioencephalitis. As such, this reaction seems to have been essentially irritative, marked by intense hyperemia and leukocytic (mononuclear) infiltration, in association with neuroglial and fibroblastic proliferation. Degenerative change seems to have been definitely of secondary prominence, by which, and the severity of the medullary involvement, differentiation may be made pathologically from acute anterior poliomyelitis as well as Landry's paralysis, rabies and tetanus. Differentiation from epidemic encephalitis, however, is extremely difficult, although, as a rule, in the latter the cortical change is less marked and the oculomotor nuclei are rarely uninvolved.

The authors direct attention finally to the similarity between the findings in their case and those determined by Marie and Lhermitte in Huntington's chorea particularly in point of involvement emphasis upon the cerebral cortex and corpus striatum, and maximally upon the latter. This, being an outstanding feature in both affections, throws into signal relief the possible significance of striate disorder in the genesis of choreiform movements so-called.

RAPHAEL, Ann Arbor, Mich.

EPIDEMIC HICCOUGH. A. HANNS, Progrès med. **36**:257 (May 28) 1921.

The nature of epidemic hiccup is still obscure but tends, more and more, to be considered as an elective and solitary localization of attenuated virůs of epidemic encephalitis.

The chief arguments raised against the identity in the nature of these two affections have been that the hiccup always, it is claimed, exists alone, and that inversely in a case of epidemic encephalitis among its so numerous and varied manifestations, the hiccup has not been observed. Such arguments are refuted, however, by the case which the writer takes occasion to report.

In this case there was persistent hiccup, more or less constant, from the seventh to the twenty-third day of an afebrile illness in which headache, photophobia, somnolence (lasting twenty-one days), paresis of an arm, facial paralysis, ptosis and dysarthria were concomitant symptoms. The hiccup involved diaphragmatic contractions, as well as symmetrically bilateral contractions of the scaleni muscles.

The diagnosis was accepted as encephalitis. The case appeared to prove that hiccup can be a part of the symptomatology of encephalitis and tends to show that monosymtomatic hiccup is a "forme fruste" of an encephalitis.

DAVIS, New York.

CYSTICERCUS SPINAL MENINGITIS. TITUS VASILIU, Presse méd. **29**: 522 (July 2) 1921.

The case described was first diagnosed as spinal cord tumor, between the seventh cervical and second dorsal segments. Necropsy revealed an enlargement of the whole cervical cord due to a grossly thickened pia, from 2 to 10 mm. in thickness. The microscopic appearance suggested tuberculosis. Microscopically the vessel walls were found markedly thickened; the intima was rich in connective tissue cells, leukocytes and giant cells ("giant-cell endarteritis"); the lumina contained plicated cysticerci. No diagnosis could be made between a *taenia solium* and a *saginata;* hooklets were not found. No worms were present in the intestine.

Askanazy (*Deutsch med. Wchnschr.*, 1902, p. 431) is quoted as having found cysticerci only outside the vessels, and Jacobsohn (*Monatschr. f. Psychiat. u. Neurol.* **21**:119) as finding the walls of cysticercus capsules made up of the walls of small vessels. Vasiliu believes that the particular histologic picture in his case is unique; besides the presence of such an endarteritis, the limitation of lesions to the meninges without invasion of the nerve tissue is noteworthy.

HUDDLESON, New York.

CONCERNING THE INTRASPINOUS TREATMENT OF NEUROSYPHILIS. Sicard and Paraf, Rev. neurol. **27**:1033 (Oct.) 1920.

Comment is made on the favorable results reported by other workers, notably Marinesco, Swift and Ellis, as attained by the treatment of neurosyphilis with autoarsphenamized serum, intraspinally administered. This seems to be somewhat contrary to the authors' experience, since they noted in a series of fourteen cases carefully observed over a long period (mainly tabetic and paretic patients) that practically no improvement followed such treatment except in cases in which the subdural schedule was combined with general intensive intravenous and intramuscular therapy and that, in addition, in this series, there occurred two sudden deaths shortly following subdural injections. The authors feel the chief value of the intraspinal technic to lie in the induction of increased meningeal permeability, thus allowing for the freer entry into the neuraxial field of medication reaching it through the blood stream and suggest as safer and more efficient in the treatment of neurosyphilis, the intraspinal administration of simple sterile saline (0.7 per cent. in amounts of 4-10 c.c.) in intensive association with general intravenous and intramuscular therapy.

Raphael, Ann Arbor, Mich.

EPIDEMIC ENCEPHALITIS AND PREGNANCY. Marinesco, Bull. de l'Acad. de méd. **85**:34 (July) 1921.

Two cases of pregnancy, complicated by epidemic encephalitis, show that pregnancy and delivery appear to render the encephalitis more serious.

The apparition of epidemic encephalitis in the course of pregnancy exercises an influence on it comparable to that of most infectious diseases. In some instances, the pregnancy can complete its course and the new-born remain free from any contamination. In other instances, the encephalitis exercises an unfavorable influence and brings about an interruption of the pregnancy; an interruption probably due to a direct intervention of the virus by way of the placenta.

In two cases (one reported by Harris and one by Novals and Sousa) of encephalitic symptoms in new-born children, the disease was very light, for it was over at the end of a few days. This argues an attenuated virus.

This attenuation of placental transmitted virus makes it seem possible that when the death or expulsion of the fetus occurs, factors other than placental contamination, such as prolonged hyperthermia and disturbances of metabolism metabolism are also at work.

Davis, New York.

THE SACHS-GEORGI REACTION FOR SYPHILIS. A PRELIMINARY REPORT OF MORE THAN ONE THOUSAND COMPARATIVE WASSERMANN AND SACHS-GEORGI TESTS. S. A. Levinson and W. F. Petersen, Arch. Dermat. & Syph. **3**:286 (March) 1921.

The paper gives the technic of this test and discusses it in comparison with the Wassermann. For the technical part the reader is referred to the original. The authors state that in an examination of 1,042 serums and cerebrospinal fluids by both the Wassermann and the Sachs-Georgi reaction there was an agreement in 92 per cent. In sixty-two cases, 58 per cent. of which gave positive clinical evidence of syphilis and in which the Wassermann reaction was negative, a positive or doubtful reaction was obtained by the Sachs-Georgi method. The conclusions reached are that the technic of the Sachs-Georgi is more simple than the Wassermann test, more uniform results are obtained and in routine work it is of invaluable aid when done in conjunction with the Wassermann test.

Patten, Philadelphia.

HEAD TRAUMA. H. Billet, Progrès med. **36**: 323 (July 9) 1921.

The writer believes that there should be systematic surgical exploration of every scalp wound and trepanning if the exploration shows even slight solution of continuity of the cranial wall.

In states of trauma without scalp injury, most careful examination of the cranial vault for depression and examination of the patient for any signs of localization are necessary, as well as lumbar puncture in all instances. Do not fear taking an appreciable quantity of spinal fluid, even from 30 to 50 c.c. Stop only when the liquid no longer flows with pressure.

If the removal of the spinal fluid aggravates the symptoms, it indicates that a double subtemporal trepanning is advisable. If the condition rests the same or improves under lumbar puncture, it is advisable to hold to these, repeating them either once or twice daily, until the condition is ameliorated.

Davis, New York.

A FOCUS OF CEREBRAL SOFTENING IN THE COURSE OF ACUTE TUBERCULOUS MENINGITIS. L. Lortat Jacob and R. Turpin, Progrès méd. **36**:357 (July 30) 1921.

The authors detail these clinical facts: In a man aged 57 with pulmonary tuberculosis, there developed neck rigidity, Brudzinski and Kernig signs, fever, depression and subsequently somnolence. The spinal fluid conformed to that of tuberculous meningitis. In the third week of these meningeal symptoms, twenty-four hours before death, pyramidal tract signs appeared on the right side.

Necropsy showed that apart from the manifest lesions of the tuberculous meningitis, there was an area of subcortical softening of the left cerebral hemisphere. The writers review briefly the subject of tuberculous involvement of encephalic vessels and conclude that in their case the focal lesion found was due to a local tuberculous endarteritis.

Davis, New York.

Society Transactions

NEW YORK NEUROLOGICAL SOCIETY

Three Hundred and Eighty-Ninth Regular Meeting, May 3, 1921

Foster Kennedy, M.D., *President, in the Chair*

RESOLUTIONS ON THE DEATH OF DR. HYMAN CLIMENKO
1873-1921

Hyman Climenko was born in Russia. He received his preliminary education in that country, having attended the "Gymnasium," a secondary school, up to the time he emigrated to America in 1890. Economic necessity forced him to discontinue his studies for a time, but subsequently he resumed them and entered Long Island Medical College in 1900, being graduated from that institution in 1904.

After a period at the Montefiore Home he went into general practice, but kept up his neurologic studies by associating himself with Dr. Joseph Collins at the New York Post-Graduate Medical School and Hospital, and later at the Neurological Institute. About ten years ago he was appointed adjunct visiting neurologist to the Montefiore Home, later chief of clinic at the Mt. Sinai dispensary, and shortly before his death adjunct neurologist to the Mt. Sinai Hospital and full visiting neurologist to the Montefiore Home and Hospital for Chronic Diseases. He was a member of numerous medical societies, among them the New York Neurological Society and Academy of Medicine, and two days before his death was elected chairman of the Section of Neurology and Psychiatry of the Academy.

Dr. Climenko's death came as a shock to his friends. In perfect health, he was taken ill with acute abdominal pain. The condition was diagnosticated as acute intestinal obstruction; he was immediately operated on but he failed to rally and died within three days from a severe toxemia. He is survived by his widow and five children.

Hyman Climenko was an able physician and an excellent neurologist. He was rapidly rising when his untimely death put an end to the fond hopes of his friends and his own aspirations. Since 1908 he had contributed forty papers on neurological subjects. A short list of titles includes Anterior Poliomyelitis, Familial Optic Neuritis, Tumors of the Brain and Cord, Dystonia Musculorum, Infantilism, Syringomyelia and Leprous Neuritis, Corpus Luteum in Neurological Practice, and Lethargic Encephalitis. His work combined scholarship with accuracy of observation.

Hyman Climenko was a most lovable man. He had a big heart, was a true and devoted friend, literally had no enemies, and was ever kind and considerate. He was so modest that he never made a strong first impression, but close acquaintance quickly revealed the sterling man. Beside his medical activities he showed great interest in liberal social and literary movements. In him the New York Neurological Society has lost a valuable member and an unusually diligent and industrious worker.

I. Abrahamson,
I. Strauss,
I. S. Wechsler,
Committee.

Dr. Foster Kennedy wished to say he had enjoyed knowing Dr. Climenko. He was a gallant fellow, a happy fighter against large odds and an intellectually honest man. Climenko possessed also a quaint and whimsical humor, and enjoyed nothing better than to be lightly chaffed himself. He was a very lovable and a very real person, and Dr. Kennedy felt that the Society had suffered a great loss in his untimely death.

A PSYCHOGENIC STUDY OF EPILEPSY IN A CHILD. Dr. L. Pierce Clark.

Dr. Clark said that since epileptoid seizures, observed in psychoneurotic patients as early as the time of Westphal, have been favorably influenced by psychotherapy, the same method has been tried on essential epilepsy. Essential epilepsy, however, is not a neurosis but is organic in origin, and the results have not been good. Nevertheless, cases appear in which psychogenic factors so clearly overlie other causative agents that the question arises whether these trends may not be of prime etiologic and therapeutic importance.

The personality makeup of the epileptic child removes him so far from natural development that ordinary adjustments are impossible. Coincident with grand mal seizures many epileptic patients show psychopathic episodes. While these are related to their defective personalities as a whole, they are not directly related to the seizure episodes themselves, yet when these are immediate forerunners or may in themselves constitute imbricated attacks or petit mal, they are of greatest moment.

The case described is that of a boy, now 5 years old, who at the age of 2½, eight months after the birth of his younger brother had attacks which were for the most part conscious. He began to clench his fist and grimace, ending with laughing and crying. A year later the symptoms became more severe until the attacks were not only unconscious but ended in classic grand mal. A very active child, nervous and irritable from birth, of neurotic and high strung parentage, he from the first was jealous of his brother. Two types of attacks were noted, one the ordinary grand mal. About two thirds of his severe seizures were preceded by laughing or puffing attacks. The boy suddenly stood quite still and breathed or puffed as one might on entering a cold bath. The lips were drawn back and upward giving a leering expression. The smiling contraction of the lower part of the face had the suggestion of animal snarl. The eyes had a vacant, fixed, far-away gaze; the pupils dilated and were unresponsive to light for the eight or ten seconds of the attack. On one occasion when put to bed after several puffing attacks he had two minor attacks in rapid succession following displeasure. When he wanted to get up and was restrained, he threatened to have another. Instead of the usual puffing attack he merely leered and threw his arms and legs about in utter abandon. The following night while asleep he began drawing in his breath in stertorous gasps; his limbs were in tonic spasm, then clonic, and finally movement ceased as in a fairly severe grand mal. The latter would seem to have started as an ordinary puffing attack and gradually shaded over into a moderately severe grand mal. He did not waken after the attack until morning when he felt quite well. Two nights later he had a complete grand mal attack which also began as a respiratory or leering episode. A few weeks later, when annoyed, he simulated an attack, but it was a poor imitation and he admitted the hoax. Tantrums and an increasing number of nocturnal attacks, both puffing and grand mal, together with wishing spells of very childish nature, followed.

Since he was showing increased fatigue, irritability, and more attacks both mild and severe, it was concluded that his effort at attention in school was too intense and he was placed on a less taxing routine. Improvement was at once apparent, but whenever any unpleasant task was imposed he would start puffing and blowing. He admitted that he encouraged the attack by these actions, that this as well as the peculiar attack itself made his "stomach light" as though he were "floating." He would not say the acts were pleasurable, but said they gave him relief. After a time his school work was good, and he did well in his daily routine. He said he did no more puffing now "because things were going all right and he didn't need to." Whenever the routine became irksome the tantrums or attacks or wish episodes, would occur. Apparently the conscious puffings were employed to while away the general tedium of life or displeasurable states that were to be abreacted. The act was usually conscious at its initiation and if persisted in became an unconscious laughing crying attack and, if this was not controlled or aborted, after two or three of them had occurred the whole episode might end in a classic grand mal.

Remarkable improvement has been noted in the past four months. Tantrum episodes rarely appear, and evidences of a desire to retreat from reality in his wish fantasies and in puffings are eliminated; the grand mal has been absent now for over four months. The retreat, or displeasure mechanism, was apparently first employed at the birth of his brother when his mother's attention was diverted from him. The boy shows the grand mal epileptic in the making, and that the disorder is based on a defective epileptic anlage of constitutional origin. The personality uses the defective instinct of adaptation for its own ends—a conscious and unconscious retreat from reality, which is proportionate to the stress demand.

DISCUSSION

Dr. Smith Ely Jelliffe said that he agreed absolutely with Dr. Clark. Observation from day to day, training and morale adjustments, all are important, and we should be in a better position to treat the kinds of attacks that are usually lumped together under the title of epilepsy.

Dr. Clark said that the personality of the little boy had undergone a remarkable change in the course of his observation. His physiognomy and voice, manner of walking, have all changed. It was thought previously, by his family, that he was not so different from other children, but with his improvement they are surprised that they did not see his shortcomings before. The difficulty now encountered is that of injecting the boy into his home environment when his course of treatment is completed. Transplantation into a different family may be necessary for a number of years. Even though the relatives might be trained and think that they understand the situation, they would nevertheless react to family type.

EPIDEMIC ENCEPHALITIS SIMULATING BRAIN TUMOR. Dr. Irving J. Sands.

These cases from the Psychopathic Service of Bellevue presented all the general signs of brain tumor, such as headache, choked disks, hemiplegia and vomiting of the projectile type. In two of the cases operation had been decided on but permission for operative interference was refused, in one instance by the patient herself and in the other by the patient's parents. The third case terminated fatally.

Case 1, a boy of 12, of negative family and personal history, in June, 1920, began to show loss of interest in his surroundings, with awkwardness in the use of the left hand. Then followed a steadily increasing paralysis of the left side of the body which later spread to the right side, with choked disks, first in the right and later in the left eye. The paralysis was at first flaccid and later spastic. He lost control of his sphincters and had dysphagia at first for solid and later for liquid food. The mental picture was that of indifference, but the patient responded to questions, and his answers were relevant and coherent and his emotional reactions adequate and consistent. Serologic examinations excluded syphilis. Permission for operation was refused by the parents. The patient began to recover in the middle of September, 1920, and was discharged from the hospital in the middle of November, 1920. He was last seen in March, 1921, had returned to school and was getting along fairly well except that he was quite readily excited and irritated and would fly into rages. Physically he showed the residuals of an old left hemiplegia, with slight haziness of the right disk, and there were definite involuntary choreo-athetoid movements in the fingers and hand of the left side on voluntary motion of any other part of the body.

Case 2 was that of a 42-year old married woman of negative family and personal histories. In the middle of February, 1921, she began to complain of general malaise, headache and a slight cough. On March 1, she suddenly took to bed, refused to talk and would see no one. In the hospital she showed evidence at first of right hemiplegia, with choking of the left disk. Later the process extended to the right side of the brain producing greater choking in the right than in the left disk, with hemorrhage in both disks. Subsequently she showed considerable meningeal irritation. Serology was negative for syphilis. A roentgenogram of the skull was negative. The mental picture was that of steadily increasing drowsiness, lack of attention and inability to answer questions. She lost control of the sphincters and regurgitated even liquid food. She died on April 14. Necropsy was refused.

In Case 3, the patient from the very beginning showed evidence of a growth in the right side of the brain, causing left hemiplegia with elevation of the right disk. Serology excluded syphilis. She had periodic attacks of headache, nausea and projectile vomiting. Her mental picture was characterized by marked irritability and confusion. The physical signs pointed so strongly toward a cerebral neoplasm that epidemic encephalitis was not even considered as a possibility, and she was transferred from the psychopathic service for operative interference. She refused permission and began to improve. After ten weeks she showed practically no residuals of her illness.

Dr. Sands referred to the works of Bramwell, Barker, Tilney and Howe, as evidence that changes in the disks in cases of epidemic encephalitis were not usually encountered, and choked disks were indeed rare findings in this disease. He explained the choked disk on the theory that there was pressure on the supra-optic canal of Tilney, rather than by the usual theories—such as the toxic theory of Leber or the mechanical theory of Schmidt-Rimpler.

DISCUSSION

Dr. Smith Ely Jelliffe recalled a case which he had considered one of brain tumor until Dr. Kennedy pointed out to him that it was epidemic encephalitis. In still another case the picture was one of fractured base of the skull. The patient had fallen on the first day of the illness, blood was found on cerebrospinal puncture and the physicians were sure of fracture. The illness pursued a regular encephalitis course and gradually cleared up.

Dr. I. Abrahamson thought that the course of the disease was essentially different in encephalitis and in cerebral neoplasm. Even the papillitides in these conditions had different onsets and different rates of progress. Left hemiplegia in a right-handed person with aphasia indicated multiplicity of the lesions.

In encephalitis, mental signs were common, quite marked and occurred early in the disease. In brain tumors they were less evident and usually were found late in the disease or when much internal hydrocephalus existed, the exceptions being chiefly temperosphenoidal neoplasms. He cited the case of a girl aged 12 years, with optic neuritis, paraplegia—at first left and later right sided; at first flaccid, then spastic, then flaccidity plus tremors, coincident with return of power in the extremities. There was bulbar involvement; the Wassermann reaction of the blood was + + + +; the spinal fluid was negative. Epidemic encephalitis was finally diagnosed.

Another case was that of a young woman with markedly choked disks from 4 to 5 diopters, hemiparesis, ocular signs, etc. She gave a history of an infected pigmented mole on the left leg which had been removed; then a gland had been removed from the left groin, diagnosed as a melanocarcinoma. The differential diagnosis of metastatic carcinoma of the brain and an epidemic encephalitis was decided in favor of the latter; this was confirmed by necropsy.

The importance of the mental states of patients with epidemic encephalitis had to be constantly borne in mind in making a differential diagnosis.

Dr. Foster Kennedy remarked on the interesting feature of the papilledema, its suddenness and lateness in the course of the disease. It appeared over night. The maximum was reached in twenty-four hours. Such speed of progress is not seen in any other condition. The vision was obscured with scotomas. It had not been easy to discriminate between tumor and encephalitis.

The type of encephalitis described by Dr. Sands is new, though it is not appearing frequently. It is often associated with infective states. It seems to be a phenomenon of these times, and is possibly a condition following the influenza epidemic.

Dr. Sands, in closing, said that while the clinical pictures of epidemic encephalitis that have lately been reported are quite different from those reported two years ago, one would not hesitate to declare them to be caused by the same organism. The mental picture is a great aid in arriving at the correct diagnosis. The apparent symptoms must be differentiated from the real ones. While these patients seem to be in a state of lethargy, on closer observation they are often quite keen and responsive to questions. In brain tumor cases, on reaching such an apparent or real state of lethargy, the patient would never be able to respond to questions and to cooperate for an examination as readily as in cases of epidemic encephalitis simulating cerebral neoplasms.

THE SIGNIFICANCE OF THE REPORTED NERVE CELL ALTERATIONS IN DEMENTIA PRAECOX (WITH LANTERN SLIDES).

Dr. Clarence O. Cheney.

The speaker called attention to the nerve cell and other cortical changes in cases of dementia praecox, as described by various observers, particularly Mott, who has maintained that the nerve cell alterations were responsible for the clinical symptoms of this mental disorder. With the desire to determine the validity of such claims, the speaker had undertaken histologic cortex studies of a series of dementia praecox cases, with another series of manic-depressive cases for control, using photomicrographs extensively. The comparative cortical appearances in the dementia praecox and manic-depressive cases were demonstrated by lantern slides. Conclusions were reached to the effect that in cases of long-standing dementia praecox a nerve cell loss was not evident as compared with cases of manic-depressive psychosis of similar age and cause of death; that frequently identical nerve cell changes were found in manic-depressive cases and cases of dementia praecox; that a marked variability in the nerve cell appearances was present not only from case to case but in different microscopic fields of the same case; that there was no uniform or constant cortical or individual nerve cell picture in dementia praecox which would enable one to distinguish such a case from one of manic-depressive psychosis. It was considered as unproved that the nerve cell changes found were essential to the clinical picture of dementia praecox as it could not be definitely stated how much of these changes might be due to antemortem physical disease, postmortem alterations or technical procedure. It was stated that the study had emphasized the importance and necessity of obtaining dementia praecox brain material which had not been affected as far as could be known by acute or chronic physical disease, and which was as free as possible from the objection of postmortem change, and of comparing this with similar material from normal persons. It was felt that until positive results should be found in such dementia praecox material as compared with normal material we would hardly be justified in looking on dementia praecox as an organic brain disease.

Book Reviews

THE MANNER OF MAN THAT KILLS; SPENCER-CZOLGOSZ-RICHESON. By L. Vernon Briggs, M.D., Director of the Massachusetts Society for Mental Hygiene, Member of the American Institute of Criminal Law and Criminology, Société Medico-Psychologique of Paris, France; New England Society of Psychiatry, American Medico-Psychological Association. $5.00 net. Pp. 440. Boston: Richard G. Badger, Publisher, The Gorham Press.

This is a study of the life history of three famous criminals. They are famous because their crimes were picturesque, attracting a great deal of local attention in the case of two of them and in another national notoriety because the victim was the President of the United States. It is these adventitious circumstances that give special interest to these histories; in their essence and in the genesis of the crime they do not differ from the ordinary variety of criminal records.

The book is published primarily for the purpose of emphasizing the mental and nervous defects in these murderers that the public may be stimulated to renewed efforts to search out the defective and so far as possible segregate and treat him. This is the aim of mental hygiene everywhere, but unfortunately it is a task so difficult and of such enormous proportions that little headway can be made. This book will be of most value to the jurist and to lay persons who need to study the life histories of such defectives to get the proper point of view. It will be of less value to the psychiatrist who meets these cases in his daily work.

The most picturesque of the three cases is that of Bertram D. Spencer, who was executed Sept. 17, 1910, for the murder of Miss Blackstone. It would be difficult to find in criminal annals a more striking career than that of Spencer. The family history is replete with insanity and nervous diseases of one sort and another. His father was a man of ungovernable temper and frequently punished Spencer in a cruel and inhuman fashion. As a boy he was subject to attacks of ungovernable anger. These attacks persisted into adult life and were brought on by trivial situations. For the most part he was affable and was kind to his wife and child. Approximately two years before the murder a number of burglaries occurred in Springfield, Mass. Fourteen of these crimes were definitely traced to Spencer. They were all committed in about the same way. He would enter a house, not necessarily when the inmates were asleep, but walk boldly in and insist on their giving him some money and then would ransack the house for valuables. The lower part of his face was always concealed by a dark handkerchief. He carried a revolver and threatened the inmates. He seemed to prefer being seen while he took enough money and some valuable articles. He would frequently pass by things of great value to pick up objects of little or no worth, such as collar buttons and small articles of jewelry. After he was apprehended more than one hundred such articles were found in his home. His last burglary resulted in the killing of Miss Blackstone, for which he was finally apprehended, tried and subsequently executed.

A difference of opinion as to how Spencer should be classified would be pardonable. Some might call him a psychopathic personality. He certainly was of subnormal intelligence, but how low he was in the intellectual scale we do not know, as there were no formal tests made of his intellectual stature. Perhaps that midwestern diagnosis of "feeblemindedness with a touch of praecox" would come as near a description of Spencer as any other term.

At his trial abundant evidence was introduced showing his defective heredity, want of self-control, and competent witnesses testified that he was irresponsible. This resulted in his being sent to the Bridgewater Asylum for observation. The monthly reports that were sent to the justice who committed him for observation described fully his defects with repeated opinions that he was insane. After having been under observation for more than a year, the superintendent seems to have discovered that there was a difference between medical and legal insanity, so that he then certified that he was "legally sane." Accordingly he was remanded for trial.

The least interesting of the cases is that of Czolgosz. There is a full description of the methods which were employed to facilitate Czolgosz' journey to the electric chair. There is little question that Czolgosz was suffering from dementia praecox. The disease began about three years before he killed President McKinley. This was rumored at the time, though it was generally claimed that Czolgosz was an anarchist. There is little evidence that he ever was an anarchist, and both he and his crime were disowned by that cult.

From a psychiatric standpoint the case of Richeson has the greater interest. It would be difficult to make a diagnosis of his defect in the absence of a thorough psychic analysis. Richeson was a clergyman with a rather bizarre history, but brilliant and affable. He was a man with a good education, but he had strong sexual impulses. He procured the death of a woman to whom he was engaged by administering to her or persuading her to take cyanid of potassium. All his life he had been subject to severe attacks which were variously described as hysterical, in which he would talk irrationally and incoherently and which were accompanied by amnesia.

At first he denied his guilt, and when arraigned he pleaded "not guilty." Subsequently in a letter to the prosecuting attorney he acknowledged his guilt and changed his plea to "guilty." He was sentenced to be electrocuted, and this without a trial before a judge or jury. While confined in the jail, he succeeded in castrating himself with a tin cover of a preserve jar from which he nearly died of hemorrhage.

THE CLINICAL EXAMINATION OF THE NERVOUS SYSTEM. By G. H. Monrad-Krohn, M.D. (Christiania), M.R.C.P. (Lond.), M.R.C.S. (Eng.); Lecturer in Neurology at the Royal Frederick University, Christiania, Physician to the Neurological Section of the State Hospital, Christiania, also in Charge of the Hospital's Out-Patient Department for Nervous and Mental Diseases; with a Foreword by T. Grainger Stewart, M.D., F.R.C.P., Physician to Out-Patients, National Hospital for the Paralyzed and Epileptic, Queen Square; Physician to the West London Hospital; Physician to the Central London Ophthalmic Hospital; Neurologist to the Special Surgical Hospital (Ministry of Pensions). Price, 6 shillings. Pp. 129. H. K. Lewis & Co., Ltd., 1921.

This opusculum of only about 3,500 words (excluding foreword and preface) contains an astonishing amount of information. The author, a Norwegian writing English, evidently has turned his back on the polysyllabic cult. His point is clearly stated in a few short words. Verily it is restful to read English which is correct, yet aspires to be neither eloquent, elegant nor pedantic.

The field indicated by the title is exceedingly well covered, very little is omitted, and the reviewer has discovered only one misstatement: that hoary and generally credited error, that in trigeminal neuralgia the nerve trunks are tender at their points of exit. Not only are the methods of examining the motor and sensory systems, the reflexes, the gait, the special senses, etc., clearly and completely given, but electrical examination and examination of the spinal fluid are included, as well as some very practical hints on simulation. Furthermore, the appendix of twenty-one small pages covers surprisingly well the Binet-Simon tests, aphasia, diplopia and the vestibular tests of Bárány, the last described and explained with unusual clearness and simplicity. Indeed, the thoroughness of the work, considering its size, is surprising. A few unimportant omissions may be noted. The pilomotor reflex is not mentioned nor that the cremaster reflex often is best elicited by sharp pressure on the inner surface of the thigh above the condyle. While lateral deviation of the umbilicus in tensing of the abdominal muscles is mentioned, the much more important Beevor's sign (upward movement of the umbilicus in transcord lesion at about the tenth dorsal segment) is omitted, as is the rectus jerk which may be quite informative. In the category of signs distinguishing functional from organic paralysis, Hoover's sign is not mentioned. Under "Movements of the Head" the characteristic attitude and movements of spasmodic torticollis might well be noted. Probably every experienced neurologist will fail to find a few of the tests or tricks he has learned to use, but he will find a good many he has overlooked or fails to use. Perhaps in future editions the author will take a little more space to indicate the significance of a few more of the signs and symptoms.

This little book will fit into a coat pocket or a hip pocket. It is good reading for a motor car, a street car or a railway car, and if a few thousand practitioners and a few hundred young neurologists of this country would commit its contents to memory, the American neurological level would rise like the Wissahickon in a spring freshet.

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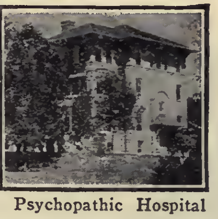
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Archives of Neurology and Psychiatry

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SOME THEORETICAL AND SOME PRACTICAL ASPECTS OF PSYCHANALYSIS *

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PHILADELPHIA

Is psychanalysis in part or in whole a form of mysticism or semi-mysticism? The affirmative of this question is strongly .resented by most psychanalysts.

I may fail to apply academic logic to my consideration of psychanalysis, neglecting references to secundum quid, tertium quid and ambiguous middle. With regard to the last, both the defenders and the opponents of psychanalysis not infrequently have shown weakness and confusion both fore and aft as well as in the middle. Let us first see what the psychanalysts say of each other.

One of the first to challenge the hypothesis of Freud as savoring of mysticism was Jung.[1] "It must be admitted," says Jung, "that Freud's school makes use of the conception of libido which passes beyond the bounds of its primary definition. Indeed this must produce the impression that one is working with a mystical principle."

Jung, who thus challenged Freud, shows himself at times to be a disciple of the occult. Witness, for example, the manner in which he uses the symbolism of numbers. The use of numbers in symbolism by the psychanalysts naturally recalls those followers of occultism who are known as numerists or vibrationists, of whom it is sufficient to say that to the numbers from one to nine very special significance is given. Number one, for example, has a vibration from the sun; number two from the moon; number three has a larger ray of symbolic significance besides the psychanalysts' directly phallic interpretation; five has the strongest psychic vibration.

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* Read at the Forty-Seventh Annual Meeting of the American Neurological Association, Atlantic City, N. J., June, 1921.

[1] Jung, Carl G.: The Theory of Psychoanalysis, Nerv. & Ment. Dis., Monograph Series, No. 19, New York, 1915.

While Jung has seen fit to call Freud a mystic, on the other hand, Ernest Jones,[2] a close follower of Freud, says of Jung's conception of the unconscious that it is founded on pseudophilosophy irreconcilable with science. Jones holds that Jung's views are characterized by a repudiation of determinism. He puts Freud with Huxley and Darwin and, briefly stated, calls Jung a religious mystic.

The faulty, fantastic or mystic character of psychanalysis is emphasized by certain prenatal references in its literature. Ferenczi,[3] for example, mentions one case in which he had partial or temporary success with hypnosis which he believed revealed a so-called mother complex, but later, by the use of psychanalysis, a father complex was also revealed. He says, regarding this case, that there was "at the bottom a symptom of unconscious phantasies connected with infantile ponderings on the menstruation and gravidity time periods with his mother, and amongst others on the idea of his own situation in the womb and at birth."

One might recall the "tunnel-window case" in which a ploughed field comes into view, recorded by Freud[4] and cited by Maeder[5] and others.

The symbolic interpretation, according to Freud, is that of the intra-uterine fetus spying on his parents in the act of coitus.

In the same category with these assumptions of Ferenczi and Freud belongs Clark's[6] libido conception of the origin of epilepsy with its infantile and prenatal references.

I do not know from anything I have read whether Adler[7] has accused other advocates of psychanalysis, or has been accused himself, of mysticism. It looks, however, as if his special theory of organ inferiority and psychic compensation—he being a sexual psychanalyst—has been developed as a sort of defensive reaction against being considered a mystic himself. At first sight his theory is somewhat appealing to those who tend to look for organic causes, but after well presenting his case in a series of most instructive arguments and illustrations, he with one sweep accedes to the sexual causation and interpretation of various forms of neuroses and insanities. He confirms this interpretation of his position by bringing into the discussion what is so much beloved by psychanalysts, the one legged, one handed, one eyed, dumb and lame hero myths of occult literature in whose deeds of blood and sexuality, as in his patients, he sees psychic compensation for physical inferiority.

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[2] Jones, Ernest: Why Is the "Unconscious" Unconscious? Brit. J. Psychol. **9**: 247 (Oct.) 1918.

[3] Ferenczi, S.: Contributions to Psychoanalysis, authorized trans., Dr. Ernest Jones, Boston, Richard G. Badger, 1916.

[4] Freud, Sigmund: Three Contributions to the Theory of Sex, Nerv. & Ment. Monograph Series, No. 7, New York, 1916.

[5] Maeder, A. E.: The Dream Problem, Nerv. & Ment. Dis. Monograph Series, No. 22, 1916.

[6] Clark, L. Pierce: Epilepsy a Manifestation of the Imperfectly Developed Libido in Persons of a Psychosexually Inferior Makeup, New York M. J., Feb. 27, 1915; Am. J. Med. Sc., November, 1919.

[7] Adler, Alfred: Study of Organ Inferiority and Its Psychical Compensation, Nerv. & Ment. Monograph Series, No. 24, 1917.

In MacCurdy's[8] pamphlet on the "Ethical Aspects of Psychoanalysis," we see here again what appears to be a defense against accusations of the possible mystic or at least illogical position of psychanalysis. MacCurdy says that "considering the trifling degree of correlation as yet established between structural alteration and brain function, these investigators are in the position of men, so convinced that all water comes from the clouds that they will not think of damming the river in a flood."

Is it true that the correlations which have been established during the last fifty years between structural alteration and brain function are so trifling, after all, in the light of what we have learned through anatomy, morphology, physiology and clinicopathology, regarding the cerebral representation of motion, sensation, tonicity and speech, and even of emotion and concrete conceptual and abstract thinking? Are these not worth more than all the purely psychic speculation regarding mental phenomena?

It will be recalled by those who have read the book of Kempf[9] that this psychanalyst may be said to harp on one or two strings throughout his entire volume of nearly 800 pages. The reader is reminded through chapter after chapter of the strivings of segments against the ego. The impression is conveyed that with regard to such questions as emotion, sexuality, and the like, the personality is entirely dominated by the segmental visceral system. The author makes use of the excellent experimental researches of Cannon but in considerable part at least misapplies them. To such an extent is this done that one almost gets the impression that for Kempf the mind is resident in the spinovisceral segments of the nervous system. It is a sort of recurrence, as someone has said, to the so-called abdominal brain idea of personality.

The ego assumed in Kempf's work is a rather mystical being. The author seems to deny anything like a mind or soul or indeed an active controlling brain, and uses a sort of verbal or assumed ego to help him out of many manifest difficulties.

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8. MacCurdy, John T.: Ethical Aspects of Psychoanalysis, The Johns Hopkins Hosp. Bull. **26**: No. 291 (May) 1915.

9. Kempf, Edward J.: Psychopathology, St. Louis, C. V. Mosby Co., 1920.

Kempf, more than he intends, seems to make an autonomic segmental subdivision of our organisms the controlling power. He gives us no clear idea of what he means by consciousness and seems to believe that it is in some way the expression of the entire personality of the individual—whatever that means. He would have us think with our muscles, control with our glands and viscera, and would make almost all psychotic diseases simply a struggle between affective cravings of segments and the hypothetical ego. All this is very fine, but, after all, does it get us anywhere in the final shakedown of theories relating to mind and matter?

Being a freudian, Kempf, of course, lays tremendous stress on the sexual elements in the analysis of cases. All through his book he brings out illustrations wherever he can of some boy or girl or woman or man having struggled with sexual tendencies which have more or less conquered his ego, or with which his ego has been struggling.

One can easily award a certain amount of credit to Kempf for collecting a large number of cases from St. Elizabeth's Hospital and other sources which illustrate the fact that sex or sexual ideas are often prominent in the minds of the insane, but this proves little or nothing in substantiation of his main position. Over and over again it is evident, although he speaks at times of evolution and that sort of thing, that the highly developed or highly involved human brain plays only a secondary part in the pathogenesis of the psychoses or neuroses with which he is dealing. Like most freudians he is suggestionally fascinated with his views of psychology and psychiatry. Sex is glaringly exhibited to him on every psychiatric canvas. In the book there is an extraordinary disregard of all the neuro-anatomic, neurophysiologic and neuropathologic work on the brain which has been done in the last half century.

I cannot refrain from making brief allusion to the pictures with which Kempf illustrates his volume, or rather to the legends by which he has described these illustrations. These legends furnish another example of the manner in which the psychanalyst reads into any subject the ideas which for the time being are dominating his mentality. The manner in which in one of the interpolated pictures a chasm is made to symbolize the vagina, its bordering cliffs the thighs, the foliage in the background, pubertal hair, a shadowy stream for which a boat containing a standing figure in white is heading, the amniotic fluid and all the rest are indications of the way in which the advocates of psychanalysis can be made to misuse what purports to be sexual symbolism. I cannot take the space to refer at any length to the manner in which Kempf and others have seen fit without warrant to read into paintings and statuary and much else their own ideas as to symbolism. If Michael Angelo or some of the other great worthies in art could be summoned from the astral plane to which they have been ascribed by another mystic cult, they would indeed be amazed at the meanings, never dreamed of by them, which have been given to the details of some of their great works.

Sherrington has clearly shown that an animal whose cord has been completely transected, will definitely respond with emotions like rage and fear although the animal may not exhibit some of the local phenomena of such emotions. I have seen many cases of high cervical lesions, a few complete in one or in all spinal systems, in which the power to respond to emotion both psychically and by facial and ocular expression was retained.

Observations on certain cases of paralysis agitans and pseudo-paralysis agitans are interesting in connection with this question of the abolition of emotional expression with which at least in some instances goes also a reduction in emotivity. I have reported a number of cases in which emotional expression and emotion itself were disturbed as the result of a lesion of the cerebral portion of the cerebro-spino-autonomic nervous system.

Without the concept of the unconscious, recent psychanalysis would have nothing on which to stand. To define or explain consciousness is by no means easy. Even so great a psychologist as William James [10] when lecturing on the stream of consciousness, tells us that he is not prepared to give an answer to the problem on the explanatory level; in other words, he declines to define the term and confines himself to the descriptive or analytic level.

The term "awareness," sometimes used in the definition of consciousness, is not much more explanatory than the word consciousness itself.

Let us turn for a moment to the views of three different writers on psychanalysis. At the joint meeting of three British societies concerned with psychologic questions, a symposium was held, during which was discussed the rather cryptic question "Why Is the 'Unconscious' Unconscious?" Those presenting leading papers in this symposium were Maurice Nicoll, W. H. R. Rivers and Ernest Jones.[11]

Nicoll, a disciple of Jung, largely confines himself to a presentation of the views of this psychanalyst. In much of his paper he concerns himself with what he terms the "collective unconscious" by which he seems to mean the "accumulated ancestral unconscious" of a person,

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10. James, William: Talks to Teachers on Psychology; and to Students on Some of Life's Ideals, New York, Henry Holt & Co., 1901.

11. Nicoll, Maurice; Rivers, W. H. R., and Jones, Ernest: Why Is the "Unconscious" Unconscious? Brit. J. Psychol. **9**:230, 1919.

not simply for a few generations, but the unconscious of our thousands and hundreds of thousands of ancestors during a million years. To him the unconscious is not a personal but an impersonal matter. After all, this is only another and not very satisfactory way of stating the great question of heredity. One may inherit traits, trends, tendencies, capacities or incapacities, but never an isolated memory or thought.

Rivers, who follows Nicoll, disagrees not only with him but also with his own successor, Ernest Jones. The chief tenor of the contribution of Rivers seems to be that what is repressed within the unconscious is chiefly that part of the instinctive which is not useful to the developing personality of the individual. His view in brief is utilitarian. He makes use of the well-known illustration of the larva and butterfly and also of the views of Head and his collaborators, of whom Rivers was one, regarding protopathic and epicritic sensibility.

Ernest Jones assails the point of view of both Nicoll (Jung) and Rivers. He considers neither of them tenable and attacks Jung's idea of the unconscious with a big stick in a way to which I have already referred in discussing mysticism. Jones is a hedonist and accepts the pleasure-pain theory in explaining sexual repression in the "unconscious."

In another article, Ernest Jones [12] discusses what is meant by the unconscious. He first speaks of what he terms the mental theory meaning that of no mentation occurring, as when a patient is unconscious from a knockout blow. This is the sense in which some psychologists use the term. The second theory of the unconscious to which he refers is what he terms the limbo theory, which makes the unconscious a lumber room for all sorts of forgotten or discarded memories. The third form of the unconscious Jones calls the scientific view by which he means the freudian theory. After all, this freudian view of the unconscious is also a limbo hypothesis, the one point being that the sexual psychanalysts only refer matters sexual to their limbo.

In the process of my psychanalytic reading I tallied new terms until they had reached four score or nearly this figure and by no means exhausted the subject. A voluminous terminology is often a sign of weakness rather than of strength in a science or art. A somewhat extensive terminololgy is of course necessary in some of the more exact sciences like botany or physics.

In psychiatry in particular—largely because of the uncertainty of our facts and theories—classification and classifying terms have been sadly misused. The tendency is sometimes shown by men who are fond of going into minutiae, to take the insanities with well established symptoms, lesions or psychic phenomena, and divide and subdivide them until perhaps, as in the case of one of our late colleagues, we are presented with a classification which has as many varieties and subvarieties as Heinz's well-known collection of pickles.

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[12] Jones, Ernest: Papers on Psychoanalysis, New York, William Wood & Co., 1919.

In psychanalysis this tendency to division and subdivision, to the invention of new terms or the application of old terms to new uses has been carried to excess. A long list of the terms thus introduced into psychanalysis could readily be made—in fact, has been made. Each new investigator is not happy until he has invented at least a few new terms.

In the first place, we have the already considered necessary terms, consciousness and the unconscious, but various other terms have been added to these, such as preconscious, the foreconscious, the subconscious and the coconscious.

Starting with varieties of consciousness, we are then confronted with libido, fixation, repression, sublimation, regression, compensation and overcompensation, reaction, conversion, constellation and many other terms. The use or misuse of these terms needs continuously to be kept in mind in reading the contributions on psychanalysis; nor is this use consistent with different authorities. In the case of libido, for instance, Freud gave it a meaning which closely associated it with sexual instinct or desire, but Jung tells us it should have a more general energic significance, with which statement the freudians later have more or less coincided. Sexual trauma becomes sexual phantasy or sexual experience.

A few terms have particularly appealed to me as of dubious utility, such as narcissism, constellation and complexes, in all their extraordinary array. We all know the meaning of constellation as applied in astronomy, but its transfer to the domain of psychanalysis is one of the rhetorical methods of exploiting the subject. By constellation seems to be meant a group of related ideas. Why not then be satisfied with calling them a group of ideas, without invading the domain of the astronomer?

The term narcissism, introduced by Havelock Ellis, and speedily adopted by Freud, is another rather pleasant word which derives its meaning through the pretty myth of the youth Narcissus whose admiration of his own image reflected in the pool led to his eventual destruction. Around this word has been made to evolve a series of ideas relating to self-love and especially to libidinous self-love.

The method with which Socrates—at least as represented by Plato—perforated the terminologic bladders of the ancient sophists, letting out their hot air and obnoxious gasses, might equally be used in attacking the terminology of these modern freudian sophists. It could be readily shown that the large use of terms by the latter is in not a few instances defensive, serving to get them over hard places.[13]

Of all the terms which have been invented and amplified, that of complex is perhaps the most striking. It was, I believe, originally applied by Jung and his immediate followers to extricate themselves from the difficulties which arise in the consideration of ideas in the unconscious supposed to have emotional or instinctive associations. Freud, ever on the alert for new philologic weapons, after a little resistance, adopted the term complex, until now the freudians in common with those of other psychanalytic schools revel in complexes—hidden, anal, oral, castration, and finally the root or nuclear complex which has its exemplification in the Oedipus complex. The Oedipus complex and the Electra complex, especially the former, are the favorites of the psychanalyst.

Some psychanalytic writers, like Jelliffe, for instance, tell us that this Oedipus complex can be used as a sort of psychic measuring unit or foot rule. We must look, according to the Oedipus hypothesis, for some phase of the relations which the child bears to his parent of the same or of the opposite sex, of the son to the mother or father, of the daughter to the father or mother, or of the sister to the brother. Paramount in the hypothesis is the idea that these relations are in some way of a libidinous or incestuous character.

Freud, Rank, Ernest Jones, Jelliffe and others work this story of Oedipus to an extraordinary degree. Given an ancient mythologic story, a tragedy, old or more recent, a novel like Defoe's "Moll Flanders" which seems to have been rather neglected by the psychanalysts, a painting or a piece of sculpture, the psychanalyst is able to read into or out of any or all of these his preconceived hypothesis.

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[13] Since the delivery of this address in June, an article has appeared in the December to March number of the Journal of Abnormal Psychology, on "Education and Freudism," by George Humphrey, which I did not see until August of this year. In this article the author ably discusses the subject of terminology more fully than I did in my address, the difference being partly due to the fact that I had to confine my paper within certain time limits. Humphrey shows clearly the manner in which terms have been given an exaggerated importance and just as clearly states what I had in mind and briefly expressed, that the terms used in psychanalysis are often paraded as if they were actual entities instead of more probably helps to the exposition of false psychic hypotheses. Humphrey is particularly clear with regard to the use of such words as conflict, projection, interjection, and the like, so attractive to the exponents of freudianism. These terms, he holds—and I agree with him—are given utterly unjustifiable valuation, and much of what they are supposed to illustrate can be readily explained on the principle of the conditioned reflex.

One of the first of these efforts was that of Ernest Jones to discover the Oedipus complex in the drama of Hamlet, a task in which I think he was not altogether successful, as was pointed out by Lloyd.[14]

Jelliffe[15] tells the story of a patient with various evidences of inherited and acquired imperfections and derelictions to whose complex the psychanalyst could not obtain a clue until at last he learned of a monotonous breakfast in which his patient had indulged for fifteen years, consisting of waffles and maple syrup and sausages, rather small. A further inquiry into the patient's dietary revealed the fact that for years he had lived chiefly on milk, potatoes and meats. The application of the Oedipus complex to this breakfast and to the generally restricted dietary of this dissolute young man, if not mystic, at least may be regarded as far fetched and fantastic. The milk brings him in Oedipusian relations with his mother's breast and nipples. The sausages are symbolic of the phallus and even the waffle iron is made to symbolize a pig's tits and thus play its part in carrying out the interpretation. It does not seem to me necessary to comment on this supposed illustration of the Oedipus complex, and I will leave my auditors to decide for themselves as to the good sense or nonsense of the illustration.

The study of Sophocles' tragedy is rather enlightening with regard to the extensive use of the Oedipus complex in psychanalysis. In the first place, because of the prophecy of Phoebus that the son of Laius and Jocasta would murder his father and marry his mother, his parents when he was only a few days old handed him over to a messenger, who was to take him with his limbs gyved and expose him to die in some desert place. It is to be observed that he had probably never been suckled by his mother at all. In the second place, Oedipus, whose life was saved through the kindness of a shepherd and who was adopted by the king of another country, was brought up with the idea that his adopted father and mother were his real parents. Later he became a conquering hero. On one occasion, meeting by chance his father on the highway, he slew him in resentment for what was done by the king's herald and was attempted to be done by the king. He had no knowledge that he had killed his own father. Still later he married his own mother Jocasta, the widow of the king. In other words, both the murder and the incest were accidental and had absolutely no relation to the infantile and juvenile experiences of Oedipus. It is true that the story has in it both incest and murder, so dear to the hearts of the psychanalysts, but it is utterly lacking in the main elements of the freudian idea of infantile sexuality and the child-father and child-mother complex.

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[14] Lloyd, James Hendrie: The So-Called Oedipus-Complex in Hamlet, J. A. M. A. **56**:1377 (May 13) 1911.

[15] Jelliffe, Smith Ely: The Technique of Psychoanalysis, Nerv. & Ment. Dis. Monograph Series, No. 26, 1918.

Under the lead of Freud, the symbolism of dreams has apparently become the chief support of direct psychanalysis, which without dream analysis would probably have soon fallen to the ground.

In profound slumber it is probable that we do not dream any more than we do in what might be termed the knockout unconsciousness which results from a blow. The dreams which can be studied directly in our waking hours, or which can be reproduced by special processes as by hypnosis and deep abstraction, represent what occurs in the predormitial, postdormitial or intradormitial states, that is, in states preceding or following sleep, or in a period of shallow slumber.

A few well known or elemental facts regarding dreams may here be recalled, namely, that they are dependent on experiences recent or remote; that they may be in their content either contemporaneous, retrospective or prospective. They are usually initiated by recent and often very recent experiences. Once initiated, the dream fantasies, through free associations may carry back in time and expand more or less indefinitely. The prospective or anticipatory content of dreams, as Peterson[16] has pointed out, has been largely neglected by psychanalysts in general, in spite of the fact that wish fulfilment plays so large a part in the freudian dream theory.

I have analyzed, or at least have studied, many dreams, not only those which I have experienced myself, but also those of others. In this study I have particularly made an effort to inquire into the dreams of persons of different types and ages. A few dreams are not only frankly sexual, but they may be both retrospectively and prospectively of this description, this fact depending largely on the contemporaneous existence of sexual ideas, tendencies and even experiences of the dreamer. Dreams as to hunger and thirst usually have their initiation in recent or contemporary sense experiences of the individual, or in the verbal or written recital of others.

My views also are entirely in accord with Peterson in statements like those which follow:

> The currents of dream consciousness would seem to be a kind of reflection of the currents of alert consciousness, a moonlit underworld of daily common life, with wider horizons as to past and future. . . .

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[16] Peterson, Frederick: The Psychology of Anticipation and of Dreams. Contribution to Medical and Biological Research, dedicated to Sir William Osler, New York, Paul B. Hoeber, 1919.

> It would seem as though sex and hunger play a secondary part in the behavior of mankind in general. Survival and reproduction are necessary to man's advance as conqueror of his environment, but the goal of his ultimate consummatory reaction is the encompassing by his brain and mind of the planet and the stars.

The "stars" and "planet" thus metaphorically referred to include matters intelligent, intellectual and cultural.

It is the manifest content of dreams which chiefly concerns the dreamer and should chiefly concern the analyst. Symbolic meaning may sometimes be found in this manifest content, and its vagueness may be cleared away and its limits expanded by processes of hypnosis or abstraction or perhaps by appropriate methods of psychanalysis. I do not, however, believe in the "latent dream thought" theory of Freud. To my mind it is mainly an invention of the analyst to enable him to escape from his difficulties in proving his sexual theory. The so-called latent content is in the mind of the analyst and not in that of the dreamer.

The analyst reads much into dreams by direct analysis, by suggestion and by other processes. This statement is, I know, denied by advocates of psychanalysis, but the briefest consideration will show that it has in it much of force and truth. The patient, and especially the private patient, as a rule comes to the office of the psychanalyst fully aware that the doctor is a believer in a sexual theory. The patient is in a state of mind to be led and directed and cannot fully escape from the mesh. The analyst more than the patient is the victim of a powerful autosuggestion which compels him in spite of himself to search for sexual reactions, and to read these into his analytic process.

My mind has been impressed with these ideas as to suggestion by reading many of the published accounts, usually imperfectly presented, of the cases of direct and dream analyses.

These accounts of examples of psychanalysis are usually brief, but brief or lengthy one can often find in them evidences of suggestion, persuasion and domination. In one of the latest articles on psychanalysis which I have read, the doctor, first using the method of word association and supplementing this with suggestion, persuasion and bulldozing, was able to obtain an unusually distasteful story of sexual desires and experiences from an unfortunate young woman.

The method employed in direct psychanalysis and in that of dreams is a stimulus not only to the imagination of the patient, but also to fabrication. In reading one of Jung's accounts of the analysis of a precocious girl of 11, this appeared to me quite evident. It would not be too much to say that this child had the time of her life in the dozen or more interviews with her doctor.

Only a word or two can be said with regard to phallic or sexual symbolism although this subject might easily be made one for a lengthy essay. Drawing from the stories of savage life, from folklore, from myths, from early historic writings, from paintings, sculpture and architecture, from the slang of the bowery, the workshop and the farm, from almost anything in heaven above, or the earth beneath or the waters under the earth, phallic or sexual symbols have been indefinitely multiplied. Everything elongated or capable of standing, from a banana, a snake, a cane or an umbrella, to a church steeple or the Eiffel Tower, has been described as the symbol of the phallus; and every sort of an inclosure from a work-box or a basket to a cave or chasm or cañon has been found to be a symbol of the female genital organ. It is little wonder indeed that the analyst is able to read into his examination a phallic or sexual interpretation.

Although not in France, I had during the last half dozen years some opportunities to study cases of so-called shell shock, and I had much larger opportunities of dealing with these questions in consultation with neurologic colleagues who held positions in neuropsychiatric hospitals both in France and in this country, and during both the active period of conflict and the time which has elapsed since the armistice in the fall of 1918.

To the literature of functional neuroses and insanities resulting or apparently resulting from the war, I have given much attention. As is well known, I wrote the introduction to Southard's [17] treatise of almost a thousand pages on shell shock and neuropsychiatry. In my study of this work I read the histories of almost all the nearly 600 cases which it contains. So little attention is paid in the reports of these cases to freudism, jungism and adlerism and other psychanalytic schools that psychanalysis of the sexual variety seems to have been regarded as negligible.

MacCurdy,[18] although himself a psychanalyst, in his "War Neuroses" pays little attention to psychanalytic problems.

Rivers, in his preface to MacCurdy's book, says that this author when considering the simplicity of the war neuroses as compared with those of civil practice, rightly ascribes this to the fact that the war neuroses depend essentially on the coming into play of the relatively simple instinct of self-preservation, while the neuroses of civil life largely hinge on factors connected with the far more complicated set of instinct associated with sex. MacCurdy agrees with other observers that sexual factors take a comparatively small place in the production of the war neuroses, but he believes that those who have shown a lack of adaptability to the experiences dependent on sex are also liable to fail in their adaptability to the stress of warfare.

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[17] Southard, E. E.: Shell-Shock and Other Neuropsychiatric Problems, Boston, W. M. Leonard, 1919.

[18] MacCurdy, John T.: War Neuroses, University Press, Cambridge, 1918.

The fact remains, however, that the war neuroses in their symptomatology, diagnosis and prognosis bear a close resemblance to those observed in civil practice. They are perhaps more acute and at times more severe, but they exhibit the same phenomena physically and mentally as the functional neuroses—anxiety neuroses, hysteria and compulsion neuroses—for which Freud and his followers claim that psychanalysis is the effectual treatment. The patients in the cases detailed by MacCurdy seem to have been cured chiefly by methods of powerful suggestion, rest and physical treatment, measures chiefly employed by neurologists and alienists before the entrance of psychanalysis into medical practice.

If these war neuroses, thousands on thousands in number. were developed chiefly through fear often intensified by previous fatigue, and could be speedily cured by methods not of a psychanalytic nature, why may not the milder cases with which we have always been familiar be similarly cured? Col. Robert Armstrong-Jones and Colonel Springthorpe, in a discussion before the British Medico-Psychological Association, took a strong position against the influence of sexual factors either in the etiology or in the treatment of the many cases of war neuroses which came under their observation and supervision. These gentlemen, like others who have written on this subject, show distinctly that the instinct of self-interest or self-preservation rather than any sexual experience or conditions was the great factor in producing this war neurosis which had at its base, in the vast majority of cases, fright or terror. Psychanalysis was tried in some of the cases reported by Colonel Armstrong-Jones and failed, the cases afterward being successfully treated at the Queens Square Hospital. Why waste weeks, months and years with a method of treatment not more useful than our old-fashioned methods of rest, isolation, physical rebuilding, associated with suggestion, persuasion and the personal domination of the patient by the physician?

Ernest Jones in one of his papers on war shock and psychanalysis attempts a defense of the psychanalytic views as expounded by Freud and his followers. This shrewd dialectician is not at his best in this article throughout which he seems to beg the question. Like MacCurdy, he argues that a neurosis of war can be traced in the first place to lack of adjustment or lack of adaptation plus the more immediate exciting causes of fear of exposure, and then of mutilation and death, and then of distress connected with the methods of life in the trenches. He does not pay sufficient attention to the subject of fatigue, to which MacCurdy gives proper consideration. He makes a feeble effort to relate war shock to sexual phenomena, but frankly admits that he was not able to do this to any considerable extent. He shows what every student of war psychanalysis knows, that the sexual element in the causation of the neuroses is overvalued.

Reference is made in the article by Jones to the question of the traumatic neuroses in times of peace, to the consideration of which he tells us, little attention has been paid by psychanalysts. I have had a large experience with these traumatic neuroses, having seen scores and perhaps hundreds of these cases, many of which have come to trial in courts of law, and in the study and treatment of which psychanalysis has rarely been used.

Not being a psychanalyst of the freudian or any other school, I can hardly be expected to discuss the benefits which come to the patient and the community from psychanalysis. One might as well be expected to treat of the benefits to nervous and mental cases derived from "mutilation therapy," as I am inclined to call the forms of physical treatment which have at their basis the mutilation, destruction or removal of various portions of the body, such as the teeth, tonsils, ovaries, sections of the intestines, etc. It is of course to the advocates of psychanalysis that I must leave the enumeration of its beneficial effects.

It is my belief, however, that the propaganda for psychanalysis has been of considerable benefit to physicians and psychologists in that it has directed their attention to the more minute and profound study of sex, and, as Jung would say, to other energic phenomena, although the study of sex problems has never been neglected entirely by the really able exponents of neuropsychiatry.

Some neurologists and alienists have perhaps shown a tendency to push aside or dodge sexual investigation, but I do not believe that this is true of the best practitioners in this department of medicine. They have steered a middle course between the tendency to include and to avoid sex problems, occasionally, perhaps because of their peculiarities of temperament and social development, tending to neglect the difficulties of the sexual situation.

With regard to the evils and dangers of psychanalysis: These are in the first place of a general character, although this term is not really as clarifying as it should be. They are the same evils as those which are inflicted on the profession and the community by the too intense concentration on mystic or semimystic subjects, such as spiritism, Christian science, divine healing, corcesterism, evangelical healing and the therapy of shrines, like Lourdes, Knock and Stê. Anne de Beaupré. I do not hesitate to say that great evils have come to pass from the propaganda favoring these so-called forms of cure.

To my mind it is the same evil which may come and which has come from the active propaganda of psychanalytic teachings. I do not here refer to that which has been so often expressed as the moral injury to the community, but my belief is simply that which I think every rational physician is likely to hold, that much of evil and danger may come from the advocacy and practice of any irrational form of treatment.

Like every one interested in the subject of psychanalysis, whether believing in it or not, I have given much attention to the study of the question of "transference" and the "transference neuroses." It has been defined as a feeling of acknowledged sympathy from the patient to the physician, this sympathy eventually in the course of a so-called successful analysis taking on a distinctly libidinous coloring. Almost every writer on the subject of psychanalysis, like Freud and Jelliffe, seem to recognize and discourse on the evils and dangers of "transference." The broad statement which Freud makes in connection with his discussion of this subject, namely, that "if the patient has fallen in love with the physician, there might seem to be three courses open. A permanent, legitimate relationship might come to pass, though this would be unusual. Or physician and patient should at once separate and give up the work which they had begun. The third course might not seem incompatible with the success of the treatment, namely, a temporary illegitimate love relationship."

All three of these courses are disavowed by Freud and his followers for reasons so apparent that one need not here recall them, but the very suggestion of the first and third of these possibilities is sufficient to challenge the safety of such a method of medical practice. I have known of some instances in which such dangers were realized in a serious way after the process of transference had taken place, and of course the psychanalysts themselves have acknowledged the occurrence of such untoward results. Knowing as I do that the anxiety, hysterical and compulsion neuroses can be cured in a less dangerous and less objectionable way, why should we subject our unfortunate patients to this procedure?

In conclusion I might say that I believe psychanalysis, especially the freudian or sexual variety, is tending toward the discard and in another generation will have lost its hold on the profession and the community.

A CRITIQUE OF PSYCHANALYSIS *

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I have no sympathy with the argument of ridicule which Dr. Mills has used so effectively, but, as I believe, for the moment only, and to the apparent delight of a large portion of this audience. I cannot help thinking that if Dr. Mills had confined himself more closely to the text of his prepared paper, which I had his kind permission to read in advance, his criticisms of the freudian psychology would be taken more seriously and would carry greater weight. For a serious argument of reductio ad absurdum is legitimate.

It is difficult to discuss the freudian psychology without first defining accurately what we are discussing. Otherwise we shall be discussing different things, as is commonly the case. Psychanalysis means three different things:

1. It is a method of examination of the mind for the purpose of determining what factors may be at work as determinants of any given clinical condition.
2. It is a body of doctrine.
3. It is a method of therapeutics.

When we say that we agree or disagree with, that we accept or reject, psychanalysis, we must clearly understand which of these three categories we are speaking of. Let us, then, consider them separately.

PSYCHANALYSIS AS A METHOD

Psychanalysis as method of examination of the mind by the technic of so-called "free-associations": This means simply the recovery of associated memories, of memories of past mental experiences and of behavior awakened in successive associations. Having recovered the associated memories, that is, the thoughts, wishes, behavioristic episodes and other episodes antecedent to the mental condition we are investigating, those are selected or accepted as having a causal relation to the phenomena to be explained which logically would determine these phenomena if they continued to act as subconscious proc-

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*A contribution to the discussion of the paper by Dr. Charles K. Mills on "Some Theoretical and Some Practical Aspects of Psychanalysis," read at the Forty-Seventh Annual Meeting of the American Neurological Association, Atlantic City, June, 1921.

esses and if, in consequence of conflict, they made use of certain postulated mental mechanisms by which they became transformed and disguised, as "compromise formations."

This method of "free associations" is of unquestionable value for the purpose of digging into the mind and is a distinct addition to our diagnostic technic. It has, however, its limitations and its fallacies. It cannot from its very nature, for reasons I will presently give, be relied on in itself, and particularly to the exclusion of other methods, to give exact results and therefore to permit conclusions which are unquestionable. It undoubtedly, as a method of applied psychology, gives instructive insight into the mind when conservatively and intelligently employed and permits shrewd guesses as to the causal factor for which search is made. It is one mode of attack, and sometimes the only mode at our disposal, but needs to be supplemented by other methods, particularly when the problem is to determine fundamental principles and mental mechanisms. Even for a physical examination of the body there is scarcely a single technical method that can be relied on to give absolutely reliable diagnostic conclusions: auscultation, percussion, the thermometer, the clinical symptoms, all supplement one another for the purpose of diagnosis, and the findings of each require to be interpreted. Even a positive or negative Wassermann reaction is not absolutely diagnostic of the presence or absence of syphilis. If errors attend the diagnostic technic in physical conditions and the findings of each and all methods require interpretation, how much more must this be the case with any technical method that attempts to analyze the numerous and subtle factors involved in mental motives and behavior?

The errors liable to result from the method of "free-associations" are due: (1) to the necessity of depending on interpretations and inferences to connect an antecedent experience with a present phenomenon and, for the purpose of interpretation, of postulating very debatable mental mechanisms to account for the determination by a selected antecedent experience of a present symptom or phenomenon, (2) to the fact that by the use of interpretations very different antecedent experiences can be found to be the logical causal factors of the phenomenon in question.

When in interpreting a given phenomenon, as for example, to take the stock phenomenon, a dream, we have to apply one or more of various theoretical mechanisms, such as conflict, repression, displacement compromise, disguisement (to avoid a hypothetical censor), inversion, transposition, regression, projection, introjection, transference, dramatization, condensation, sublimation, fixation, compensation, etc. —when we have to do more or less of all this in order to connect an antecedent experience logically with the dream or other phenomenon under investigation, it is obvious—at least so it appears to me—that the method falls far short of having that exactness which scientific procedure requires, and becomes a source of a large number of possible errors.

It is impossible for a method of this kind to be reliable and it must give different results according to the intellectual points of view of the interpreters and their differing personal evaluations of the data. But, curiously enough, it is by this very method that the conceptual mechanisms, doctrines and postulated principles of mental activity adopted by psychanalysis, have been "proved."

Having thus been proved by "free-associations," these postulates are then in turn employed to prove as determinants the selected associations in the psychanalysis of a given case. This seems to me, as my mind works, very much like a very vicious circle.

Here I am led to point out that in the evaluation of psychanalysis as a method we must distinguish between its use to determine the laws and mechanisms of the mind and its use in applied psychology to discover the particular origin and causes of the psychologic phenomena in particular cases. For the former purpose it is, for reasons I have mentioned, inadequate and unreliable, just as digging holes in the ground is a very inadequate method for determining the principles of geology. But just as digging holes is a very useful method for a mining engineer to employ to determine whether a gold bearing geologic formation is continued deep down in the bowels of the earth, according to geologic principles established by other methods, so digging into the mind by psychanalysis is a method of great value to obtain information regarding what is contained or going on therein. It is a method of applied science rather than of science itself. In the hands of unbiased and experienced clinicians it is capable of giving valuable results provided those results are not dependent on interpretation through hypothetical concepts and doctrines, themselves of a questionable nature. Too often the results reached are only of a greater or less degree of probability or even possibility.

But I would insist that to demonstrate the mechanisms and processes of the mind and the psychologic principles underlying its phenomena, including in the latter its aberrations, such as phobias, compulsions, hallucinations, delusions, affective conditions, stupors, amnesias, deliria, etc., and the phenomena of conflict in general, we need more exact methods, comparable at least to those employed in physiologic research and adequate to give relatively exact results.

In illustration of this point I may be permitted to cite an article by a whole-hearted psychanalyst whose manuscript I have had an opportunity to read in advance of publication. The author criticizes, and justly as I think, the psychanalysts for not having analyzed more adequately the repressing force in conflicts and having confined their attention almost exclusively to that which is repressed. He then proceeds by the method of free associations to analyze the former and arrives at the generalization that the repressing force is a wish to please the mother or father and this wish is also a sexual one. So that the conflict is between two sexual wishes and the symptoms are a compromise between them! This seems to be going some. The poor "censor" must indeed have a hard time of it!

PSYCHANALYSIS AS A BODY OF DOCTRINE

We come now to psychanalysis as a body of doctrine. We have to deal with the concepts which have been reached by the method of free associations and postulated as fundamental principles.

Let me say at the outset that there are two principles which, in my judgment, must be accepted as contributions of the highest importance to our knowledge, and for these we are indebted to the genius of Freud. One of these is the doctrine or the theory of repression and the other is that of conflict. They are intimately related, for it is through conflict that repression is induced. Before speaking of these I feel called on to say a few words about the subconscious, for this underlies and is the basis not only of the doctrines of psychanalysis but of the theories of psychopathology of every modern school of research. The theory of the subconscious is not novel, nor was it originated by Freud, though that seems to be the impression of many writers on psychanalysis. As a philosophical concept of the unconscious, of which I will later speak, it dates back as far as Leibnitz and Kant, and, later, Schelling, Herbart and Hartmann, the last having evolved it into a biologic and metaphysical system. Freud converted it into a debatable doctrine peculiar to psychanalysis. It remained for Pierre Janet, in France, and Edmund Gurney, in England, to take it out of the field of philosophical speculation and demonstrate by the inductive method its concrete reality as mental processes underlying certain psychologic phenomena. This real pioneer work, in 1886-1887, laid the basis, by the use of experimental methods, for every theory of modern psychopathology that makes use of subconscious processes. It was comparable in the field of psychology to the demonstration of radio-activity by physicists as intra-atomic processes essential for the explanation of certain physical phenomena. In using the word "demonstration" it must not be lost sight of, however, that both radio-activity and subconscious processes are logically only theories, though as theories they have attained the highest probability of truth.

Dissociation with the formation of subconscious processes that, motivated by their own emotional impulsive force, take on autonomous activity and induce psychologic and physiologic phenomena, had been demonstrated previously, as I have said, by Pierre Janet and Edmund Gurney, working independently, and their conclusions were confirmed (1889 to 1890) by Alfred Binet, William James, Max Dessoir and myself. But later Freud with Breuer (1891) discovered and applied the principle and mechanism of repression to explain the dissociation in the psychoneuroses. This although only a particular application of the dynamic principle was a marked advance. It is Freud's great contribution to our knowledge of the psychoneuroses. Its importance can scarcely be exaggerated. Nevertheless, it must not be forgotten that dissociation may be effected by simple failure of normal inhibition without repression from conflict, though this is not recognized by psychanalysts.

The motivation, however, of the succeeding subconscious processes became another problem, and the debatable doctrine of the repressed sexual wish, and particularly of an infantile sexual wish, was insisted on by Freud as furnishing an almost universal motive, or urge, impelling these processes to fulfil a sexual aim.

Without further discussion of this wearisome question, I want to point out only that the valuable theory of repression does not require the acceptance of the doctrine of the sexual nature of the conflict and of the repressed subconscious processes. That is an entirely different problem. It is to Freud's credit, too, that the basis of his investigation was the search for the "Why." No complete solution of the problem of repression, dissociation and subconscious activity and no complete understanding of the resulting phenomena can be attained without answering the question of the "Why." How and That are inadequate explanations. But while the freudian psychology has been most stimulating in directing a search for the "Why," its own answer was and is, in my judgment, radically wrong and has had a most baneful influence.

Repression, as I have said, necessarily implies and requires conflict.

The recognition of mental conflicts and their disturbing effects on the individual is as old as literature itself, but we are indebted to

Freud for the recognition of them as factors in the mechanism of repression and the important part they play in the maladjustment of the individual to the environment and the induction of the psychoneuroses. This does not mean, however, that we must necessarily accept the universally sexual nature of the conflict, or the very special mechanisms postulated through which it is conceived that the subconscious process fulfils its aim, or the symptoms are compromise-formations. After recognizing and giving due credit for the theories of repression and conflict, I must think that the greater part of the remainder of the doctrines of psychanalysis are not only unsubstantiated, but cannot be reconciled with a large body of facts which have been gained by other methods of research and which must be taken into consideration. Furthermore, nearly all the facts which have been advanced as the result of psychanalytic methods are open to other interpretations than those which the fundamental doctrines of psychanalysis postulate.

It is, of course, impossible in the time at my disposal to consider all these debatable concepts, but I may take one or two of them as examples.

Let us take the concept of the "unconscious," as used in the freudian sense. According to the true freudian psychology, a marked distinction is made between two classes of unconscious processes, namely, the preconscious, or thoughts which once were in the domain of consciousness and which can be recalled by the ordinary mechanisms of memory; and the unconscious proper which is not only considered to be inaccessible to direct introspection, but also to be essentially different in kind from the processes of the foreconscious. To reach the unconscious proper requires technical methods of procedure, such as psychanalysis. The unconscious also has marked characteristics which distinguish it from the preconscious, and it alone induces the phenomena of the psychoneuroses.

Before speaking of this I would point out that this division and distinction of subconscious processes is not only not confirmed by other methods of research, but also is distinctly contradicted by the findings so reached. Such a distinction and classification is therefore purely arbitrary and dogmatic. Although it is essential to the pure psychanalytic psychology, I think it can be clearly shown that subconscious (that is, unconscious) processes that cannot possibly be revealed, except through special technical procedure, have been antecedently conscious (that is, preconscious) and induce conflicts and symptoms of the characteristic classical kind. It is true that some latter day psychanalysts have given up this classification of the unconscious: nevertheless, we have been told over and over again that it has been "proved" by psychanalysis and is still held by Freud in his most recent publications.

As to the character of the unconscious proper of Freud, it has been stated axiomatically by an earnest group of students in Europe and America that unconscious processes are of a specific kind, have a specific origin, have specific characteristics and exhibit a specific behavior. According to this theory, the unconscious embraces only processes that:

1. Have been repressed from or kept out of consciousness because intolerable by reason of their unmoral, unsocialistic and other characteristics;

2. That for the most part never have entered awareness and therefore have been unconsciously and automatically kept apart;

3. That are essentially of infantile origin and nature, the splitting of the mind into conscious and unconscious regions "having taken place in the earliest part of childhood life, probably in the first year";

4. That involve the crude primitive instincts;

5. That are predominately sexual in character; and

6. As they, necessarily, like all processes, are dynamic, that they are sexual wishes.

To quote the exact words of one of the leading and most qualified expounders of this school, "the unconscious is a region of the mind, the content of which is characterized by the attributes of being repressed, conative, instinctive, infantile, unreasoning and predominately sexual."

I have no intention, least of all desire, to enter into any disputatious discussion of this theory. I want only to indicate the point of view from which I approach this problem and to record my dissent from any such limited theory of the subconscious. Without denying that some subconscious processes may have these characteristics (reserving an exception, as the lawyers say, to the extreme infantile origin postulated), to hold that all, or that subconscious processes generally, are of the character stated is to take a far too narrow view of the phenomena. One may say, if one likes, that he will call unconscious only those which have the characteristics mentioned and so arbitrarily define the subconscious. But that does not alter the fact that there are vast numbers of processes that are subconscious and that have not any of the six characteristics contained in the definition and particularly of repressed sexual wishes, or even of having been repressed at all, and that these take part in conflicts and repression with resulting phenomena. The statement contained in the theory is simply not true. One might as well define men (*qua men*) as criminals, or even as sexual criminals, repressed in jail, and disregard all other men who are not criminals and not in prison.

And so this conception of the subconscious is not true, not only because it is not confirmed by other methods of research at our disposition but, as I have said, it is contradicted by the findings of the latter.

The fallacy which has led to the theory is, to my way of thinking, as I have already said, due to the inexact and inadequate methods of research of which I have spoken. This doctrine of the subconscious can only be accepted by those who have not had a long training in the technic of the methods of psychologic investigation other than those of so-called "free associations" and who have not a familiarity through actual experience with all the facts as revealed. The validity of the freudian doctrine of the unconscious is of the utmost importance because on it hang all the laws and the prophets.

Let us take the doctrine of the "libido," which has been one of the fundamentals of psychanalysis, although there is a tendency today on the part of some psychanalysts either to modify it or to discard it entirely, notwithstanding that it has been supposed to have been indisputably "proved." The doctrine of the libido postulates a concept of a free energy, sexual in character, derived from the primitive sexual instinct, capable of transference from one set of processes to another, and which in its urge motivates and determines through various very special mechanisms psychoneurotic symptoms and other mental and bodily phenomena. Such a libido, sexual in character however much it may be camouflaged by philosophical considerations, is the source of all energy in the individual, though it may be "sublimated" beyond recognition in the higher moral, social, ethical and esthetic ideals and activities.

This conception of the libido entirely disregards the theories of the instincts and the data on which they are based as advanced by biologists and psychologists who are occupied in investigations in other fields of research.

The instincts and instinctive processes and the behavior to which they give rise have been the object of study to earnest students of human and animal behavior, and their investigations have presented important results. There is by no means, it is true, an agreement of interpretation, but the theories advanced are impressive and must be taken into consideration.

All this work is entirely ignored by the freudian school of psychanalysts and until recently by all psychanalysts; and yet some of the interpretations of instinctive processes derived from such studies offer explanations of human behavior which are entitled at least to consideration. None of these theories may be true but one or more of them offer explanations which are as probable and adequate as that represented by the libido.

What shall we say, for instance, to the theory of McDougall, which has engaged the earnest attention of psychologists and which finds in the urge of many instincts the motive forces which determine normal behavior, a theory by which the phenomena of conflicts can be readily explained? This theory, as well as others, is fully as dynamic as that of the freudian concept.

And if I may be permitted to strike a personal note without injecting into this discussion theories I have long advocated, I would refer to my own observations and experimental studies of conflicts and their resulting phenomena, carried on now for twenty years and more by other and, I believe, more exact and reliable methods than that of "free-association," although the latter has been freely made use of as a supplementary control. These studies have led to conceptions of motivating forces and mechanisms which wholly contradict the philosophical doctrines of the "libido" and give very different interpretations of the phenomena of conflict.

The freudian concept, it is true, makes use of the primitive instincts, but in a subordinate way, and makes the libido of the sexual instinct the predominant motivating force. The psychologic and biologic concepts, on the other hand, give to each one of them its just and equal part in the determination of mental and bodily behavior.

Again, take the doctrine of symbolisms. That symbolisms occur as mental phenomena no one questions, but the universality of specific symbolisms occurring in all minds, in all places, and in all ages, is, to my way of thinking, an extravagance which passes the bounds of inductive science. The postulation of a symbolism may be necessary to explain the connection between a mental phenomenon and some selected antecedent experience in order to interpret the latter as the causal factor, in accordance with one or other freudian doctrine; but it does not follow that the causal factor may not be some other equally well demonstrated one utilizing some much simpler mechanism. As a matter of fact, I believe that when such phenomena can be submitted to other technical methods of investigation, it will be found that in the great majority of cases we can find evidences of subconscious processes which are subject to an entirely different interpretation than that requiring a symbolism.

So I might run through the various doctrines, concepts, or hypotheses, call them what you will, of psychanalysis, and I believe that evidence could be presented that would equally well support other interpretations than those which make up the body of doctrine maintained by psychanalysis.

Similarly, one might run through the various mechanisms which have been postulated in explanation of amnesia, misspelling, miswriting, misspeaking; of misplacement, inversion, projection, introjection, transposition, etc.; and through the theories of compromise, disguise, regression, and so on, and if one should do this, I think it could be shown that although there may be a certain aspect of truth in each of them in certain specific instances, the use of them as universal formulas to be applied in every instance, not only is a fallacious method but leads to conclusions which are not confirmed by the findings by other methods of research. Amnesia, for instance, may at times be due to the repression of an intolerable memory, or by a wish to forget or some other wish; yet at other times, and probably in the great majority of instances, it is brought about by entirely different mechanisms. Thus it may be due, not to repression in the freudian sense, but rather to a neutral condition of disinterest and perhaps still more often to inhibition by another interest of greater intensity, although both are motivated by palatable aims. In other cases forces using still other mechanisms, which I cannot go into, have brought about the amnesia. But in truth the problem of amnesia is subordinate to the fundamental problem of why we remember at all.

Summing up this analytical criticism, I would say that the great majority and general body of freudian doctrines reached by this method of free-association have not only not been confirmed by other and more exact methods of research, but have also been contradicted by the results so reached.

The failure of the psychanalytic method is due to the fact that the findings require and depend on too elaborate, intricate, and, however ingenious, debatable interpretations, which themselves depend on debatable theoretical mechanisms and forces, and the method itself is not only inexact and inadequate but open to artefacts of the most subtle kind, particularly in the selection of the data. The conception of the subconscious and the theoretical structure built upon it approaches more nearly a philosophy than a science, and with philosophy we are not concerned.

PSYCHANALYSIS AS A METHOD OF THERAPEUTICS

Time will not permit me to more than touch on psychanalysis as a method of therapeutics. That cures may be effected by this method may be admitted as by any method of mental therapeutics whether of

Christian science, faith, suggestion, amulets, charms, reeducation or what not. Successful therapeutics is no proof of the truth of the concepts accepted as articles of belief. Disbelieving, as I do, the fundamental concepts of the freudian psychology, I believe the orthodox method of psychanalysis as practiced by the out-and-outers, is distinctly wrong in principle and meretricious in practice, using the latter adjective in its strict etymologic sense. In too many cases it is degrading to the personality, produces harmful results and is indefensible. Reeducation I advocate, but not of the freudian kind.

THE INFLUENCE OF THE FREUDIAN PSYCHOLOGY ON PSYCHOLOGIC AND PSYCHIATRIC THOUGHT

After having thus criticized the psychanalytic method and the validity of the doctrines, it would be ungenerous not to bear a well deserved tribute to the influence of the psychanalytic school on contemporaneous thought. I regret that the time at my disposal will not permit me to do full justice to this aspect of the subject. There can be no question that Freud, supported by a large group of enthusiastic followers, has compelled the attention of psychologists and physicians and forced them to take heed. Previously scant attention or courtesy was paid to any researches in dynamic or subconscious processes. No better evidence of the change can be adduced than this very discussion —the presentation of the topic of psychanalysis by a distinguished neurologist as the leading subject on the program of this Association today. Time was when any paper on the functional or dynamic interpretation of the psychoneuroses and allied problems was practically taboo in this society of neurologists and psychiatrists, as I can bear witness from personal experience. The older members will remember this uncultured attitude of mind. It was a resentment of new doctrines, of functional conceptions as opposed to the traditional organic conceptions—a Will to Disbelieve. The experience with the psychoneuroses in the great war has been the greatest factor in breaking down this prejudiced attitude. For, as I believe I am right in saying, there is a unanimity of agreement on the part of those who were engaged in the study and treatment of the war neuroses and psychoses, whether in England, France or America, that their pathology is to be found in dynamic, functional motivations and mechanisms and not in organic defects. This point of view and this conception are not novel, but the psychanalysts compelled attention when it was previously refused and forced the study from the point of view of the freudian psychology.

This, to my mind, is the great gain for which we must be thankful to Freud—the acceptance of the dynamic approach and the dynamic conception of aberrations of the normal personality however manifested. It is the search for the "Why" which is the final step in dynamic pathology. Undoubtedly there is at present a tendency to go too far in the search for functional causes and mechanisms and to overlook the conditions of organic disease. Sometimes one would think that there is no such thing as organic disease to consider. We must keep at least one foot on the ground and not soar all the time, or too high.

While the war forced the dynamic point of view on a reluctant profession, it has also given the coup de grace to many of the freudian concepts and in particular to the sexual wish as the universal determinant of the psychoneuroses. In other words, the freudian doctrine of the "Why"—of the libido and the unconscious sexual wish—broke down as a result of general experience with the war neuroses. Nevertheless, the main and important point is that neurologists and psychiatrists, while far from being in agreement as to the particular causal factors, mechanisms and processes that produce the symptomatic phenomena of the psychoneuroses, generally recognize that the explanation of the latter is to be found in the conflicting instinctive attitudes, strivings, impulses and reactions of human personality.

But what shall be said, on the one hand, of the mental processes of those who, motivated by the Will to Disbelieve, have steadfastly refused even to listen to any such explanations or weigh the evidence on which they are based; and, on the other hand, of those who, motivated by the Will to Believe, regardless of the canons of logic, of conflicting facts and inadequate methods of research, untrained in scientific methods of precision and without a comprehensive background of knowledge of the great store of psychologic phenomena accumulated by experimental psychology, have accepted as proved the excathedra dicta of a psychanalytic master and become devout, faithful believers in doctrines which have never at best been more than working hypotheses, if not mere philosophical speculations based on selected data? In these two types of mind we have a psychologic phenomenon equaling in interest those involved in the problems of the psychoneuroses.

When I was a boy I read in an essay by the French critic Sainte Beuve a sentence which has always stuck in my memory and which I like to think has influenced for good my mental tendencies. The subject of the essay was a modern miracle called the "Holy Thorn." The sentence ran something like this: "Some people see in this miracle a special intervention of Providence; but I can see in it only the humiliation of the human mind."

DISCUSSION ON PSYCHANALYSIS

Dr. Sidney I. Schwab of St. Louis said that there was one aspect of the subject with which he was somewhat familiar and that was the contact which psychanalysis had in the treatment of the war neuroses in the place where they happened, from the time that America entered the war until after the armistice was declared. An opportunity was given to him to test out as much of the psychanalytic procedures as could be carried out under conditions of actual service. From his personal experiences he was led to believe that much of Dr. Mills' objection to it as a therapeutic principle of general application in the neuroses was true. Dr. Schwab approached this testing without the least prejudice against the freudian psychology but rather much inclined to favor it. The war neuroses had proved the ineffectiveness of the psychanalytic method of treatment based on the assumption of its sexual etiology. In literally thousands of cases the etiologic factors had nothing to do with the instinct of sex but had a great deal to do with a more powerful instinct—that of self preservation. He stated that the experience with the war neuroses contributed this fact and there could be no denial of its importance in any discussion of the psychanalytic procedure or of the freudian psychology as applied to the neuroses.

On the other hand, a good deal of the freudian mechanism was absolutely proved to be a definite and crystallized thing in the building up, development and organization of the neuroses.

Dr. Schwab believed that these two things were well worth discussion and debate. That which the experience with the war neuroses has shown to be valuable and true should become the property of every farseeing neuropsychiatrist. The things that this experience had proved to be untrue should be discarded with all their nomenclature and with all their intricate and mystic complexity.

Dr. John T. MacCurdy of New York said that he was afraid that if the audience expected any gladiatorial exhibition from him it would be doomed to disappointment.

In following what Dr. Mills had said he endeavored to separate the various elements, and found there were perhaps three things that were to be considered. The first was definite argument, all of which was directed against psychanalytic theory in its different schools. As to that Dr. MacCurdy admitted that there was not a single objection which was not an echo, usually in different terms, but nevertheless an echo of difficulties that he himself had met in assimilating the various theories that had been put out by Freud and his followers. It was perhaps not unnatural that Dr. Mills had arrived at somewhat different conclusions than himself, considering the variance of their sympathies.

He refused to enter into the minutiae of these different points as that would take, literally, many hours and briefer discussion would be futile.

The second element was that Dr. Mills expressed certain personal opinions on which Dr. MacCurdy made no comment. He felt responsible for his own opinions and would not presume to criticize another person's opinions which were not based on an argument.

The third and most important element seemed to the speaker to be the general drift and implication of the paper, which was that psychanalysis was a rather ridiculous system of therapy or of psychopathologic theory. It was so full of logical errors as to be absolutely useless, and presumably therefore should be thrown into the discard. In fact, Dr. Mills finished with a statement that he believed that the psychanalytic movement would run its course and another generation would know it no more.

Dr. MacCurdy reminded the audience that he agreed in essence with many of the criticisms directed against psychanalysis but considered it important to realize the situation with which we are faced. The word, psychanalysis, which fifteen years ago was almost unknown, is today on every one's lips. The commoner terms of psychanalysis had become words of common speech. Patients went to physicians throwing technical terms at their heads. Psychanalysis was allegedly being practiced by laymen, and it seemed impossible to prevent this. If the majority of the medical profession viewed this spread of psychanalysis with horror, it was nothing compared to the frenzy into which it threw the more conservative men who were really interested in psychanalysis.

He thought it would be profitable to consider the actual factors that had possibly gone into the making of this situation. The first one should be recognized frankly. Psychanalysis held most of its popularity in and outside of the profession through what might be termed a pornographic lure. This was an expression not merely of the innate and perennial human interest in sex, but was also part of the general wave of unrest which culminated in the World War. There were, however, a good many evidences of people kicking over the traces of convention for some years before the war began. When psychanalysis appeared it provided a lovely pseudoscientific justification for allying ourselves with the brutes. The argument was, "We must not deny our kinship with the brutes, psychologically, any more than we do anatomically and physiologically. Therefore, if we have these instincts, we are false to ourselves if we do not express them, and express them openly"—an argument which was based, of course, on the fundamental fallacy of lack of recognition of the fact that there was such a thing as human specialization, which involved a definite inhibition of many of these instinctive tendencies in so far as they might be directly expressed.

.The second factor dealt with the mystical tendency which Dr. Mills had so emphasized, which was probably much commoner than was realized. It attracted a great many people to psychanalysis.

A third factor, of minor importance, that had something to do with its adoption as a fad, was that it provided a vocabulary with which to slang our friends. A few years ago one could not call his companion a thief or liar without causing some disturbance, but now one could talk about his complexes—a very convenient thing for a good many people. There was a large amount of this meretricious practice in the so-called Greenwich Village type of psychanalysis.

The fourth and most important factor to be considered was the actual achievement and scientific basis of psychanalysis. When Freud's psychology appeared there was a hiatus in our general biologic conception of man; Darwin and his followers had worked out his anatomic and physiologic evolution, but no one had done anything effective in studying man's psychologic relationship to the rest of animal creation and his mental specializations. Now psychanalysis, in Dr. MacCurdy's opinion, represented an attempt (he wished to stress the word "attempt") in that direction, and in so far as that attempt was sincere, in so far as it had been productive of results, there was a sound scientific basis for psychanalytic work.

He wished to raise the question of the exact medical status of psychanalysis in this connection. What did it offer as a method of therapy? As Freud and all psychanalysts had said, it was not a panacea for all psychologic ills. He thought it might be well for him to say what he considered to be the requisite for a successful analysis. The first thing is that the patient should have sufficient intelligence to grasp properly abstract ideas. The second thing was that this intelligence should have been directed, or was capable of being directed, to the pathologic nature of the symptoms and behavior of the patient. One could not cure a patient of something that he did not know he had. The third thing was that there should be a really potent desire to be well and to change the deleterious tendencies recognized by the patient. If those three qualifications were possessed, or could be fostered in a patient, he believed that such an individual could be cured and cured completely by psychanalytic procedure.

Then an important thing to consider was the "psychanalyst" himself. There were now many people calling themselves psychanalysts who were entitled to use the term because there was no copyright on it and there was no legislation in this matter, no licensing. What made a good psychanalyst? In the first place, he should be a medical man. Dr. MacCurdy doubted that he had ever had a single interview with a patient in which he did not quite consciously make use of his definitely medical education, and he did not see how that could be dispensed with. The second point was that the analyst should have a thorough psychiatric training. If one traced the scandalous stories one heard of ill-treatment in analysis (Dr. MacCurdy excluded the work of irregular practitioners, but referred to that of good ethical medical men), it would be found in practically all cases that the patient making the complaints was suffering from a definite mental disease. The analyst lacking psychiatric training had not recognized this fact, through ignorance, had talked of transference to his patient who had translated it into ideas of definite persecution at the hands of the analyst.

Dr. Daniel J. McCarthy of Philadelphia said that he was not prepared to accept the vague interpretations of symbols and symbolisms of the psychanalysts as facts, or even near facts, that one could scientifically, safely and continuously reason from to scientifically accurate conclusions. The objection to applying the freudian method to a study of the neuroses, the psychoses and psychoneuroses, is that psychanalysis, as practiced, does not lend itself to criticism by the same rules that apply to laboratory investigation in chemistry, pathology or bacteriology. What scientific workers have a right to demand is that theory should be based on facts, and the facts firmly established, dissociated from theory. Therapeutic results are no criterion of the accuracy of a theory. So much of suggestion, of autosuggestion, of the influence of the personality of the operator, creeps in, that we can no longer tell in the final analysis how important a part each plays. If such were true, Christian science or osteopathy could well point to cures to bolster up the accuracy of the theory. If in the study of a mooted point like symbolism, instead of isolated, interesting cases, a study of one thousand, or ten thousand cases of an isolated disease, like praecox, could give some uniformity of result of the dream content and its symbolization, this would approach a fact and would carry conviction. And so with the other mooted points in reference to the psychanalytic theory.

Assuming that the study of the war neuroses demonstrated that the sexual basis of all the neuroses was wrong, this did not mean that the psychanalytic theory had no value, but that it had quite as much value with only a relative percentage of cases of sex origin. The fundamental principles of repression and complex formation and conflict remained as a valuable method of study and treatment of these and other cases.

It was early demonstrated that the Ehrlich side chain theory was scientifically incorrect. Ehrlich promptly admitted this. Notwithstanding this, there never was a principle enunciated, not even excepting the Darwin theory, that did more to stimulate and activate scientific work than the Ehrlich theory. The theory, though wrong, was a tremendous stimulus to immunology and therapeutics, to an understanding of infectious processes.

The same can well be said of the freudian theory. Nothing has done more in our time to stimulate a study of the psychoses and psychoneuroses than this theory. Right or wrong, it has awakened a new interest in psychiatry and functional neurology; it has infused a new life into dying subjects. Apart from this, the great value of the freudian theory was to change psychology from an academic discussion of threshold sensations, time reactions, word associations and a philosophic discussion of the component parts of the mind to a real live study of the determination of conduct and its disorders. In medicine, it has taught us the value of properly taken histories of our patients, of the application of psychology to the interpretation of the influence of sex and sex matters in the life history of the individual. Those who have stood adamant against this theory must indeed be blind if they have not seen in their daily work many examples of the freudian mechanisms at play in the amnesias and hysterical disorders of conduct. Dr. McCarthy said that he did not see how any one who studied the psychoneuroses in private practice, in institutions and during the war could have avoided recognizing that a fairly definite percentage of cases belonged without question to the freudian conception of the psychoses and psychoneuroses; the physician who did not see this disregarded what was evident to any intelligent individual.

It appeared to him that there was an intermediate group of cases in which the sexual influence was a definite influence, but in which other physical and visceral or other psychogenic factors were at play.

There is a fairly large group of cases in which physical factors, focal infections or reactive endocrine disturbances are the cause of mental dissociation and disease, in which the sexual factor is of little or no significance.

If in studying these cases and in analyzing and classifying them, a physician or institution closed the mind to the value of the psychanalytic method and theory, a valuable aid to the approach and treatment of a very wide group of cases was lost. It did not mean that this was the only method of approach or treatment of the psychoneuroses. While Dr. Schwab, at La Fauche, used the freudian method of analysis and based his treatment on the theory of conflict at the Neurological Institute at Vichy, the treatment of the psychoneuroses was based on the principles laid down in the Mitchell school, i. e., the treatment of the human machine as a whole, giving due weight to psychogenic factors, but the various physical and visceral factors were never neglected and the reinforcement of the will, suggestion, and reeducation were employed as a therapeutic agent. The application of the principles of pathologic psychology was used from a somewhat different angle than that employed at La Fauche. Dr. McCarthy said that he thought the younger men in both groups could testify as to the efficacy of both methods.

Dr. McCarthy thought that the freudian mechanisms were not going to pass away like many of the fads of medicine of the past, but that they would become in the future a much more important, if better balanced, part of neurology and psychiatry; that the younger group of men growing up into this atmosphere could not help but be imbued with the freudian terminology and its mechanisms and pathologic mental states.

Dr. George H. Kirby of New York expressed the view that there would be general appreciation of Dr. Mills' suggestion that the time had come to undertake the discussion of the issues of psychanalysis more on their merits and less on our emotional reaction to them. Dr. Mills in his presentation spoke of psychanalysis as if it were a fixed system, with settled formulations and a definite technic—a procedure that had been in use sufficiently long for one to draw conclusions as to its real value. Dr. Kirby felt, however, that developments in this country had been far from uniform, both in the theory and practice of psychanalysis. It was true that the original formulations of Freud had been faithfully followed by a few, but, on the other hand, there had been from the first an independent attitude on the part of many who had declined to be bound by a rigid dogma. It will be found that in practically every center of psychiatric research in this country there has been a notable departure from, or a modification of, the freudian doctrine with the result that the developments of recent years show a marked divergence from the original freudian formulations. The broader psychobiologic conceptions now current in American neuropsychiatry differ not only in form but also in principle from the freudian doctrine. He stated that this had been forcibly emphasized at the last meetings of the American Psycho-Pathological Association.

The speaker did not wish to disparage the constructive value and stimulative influence of the Vienna School—this should be acknowledged by all. But it seemed to him that much of what Dr. Mills found to criticize was already admitted as untenable and to a large extent unessential to the acceptance of the more fundamental principles established through the psychologic, or more precisely the biologic, method of approach to the study of the neuroses and psychoses.

Dr. Kirby said that pansexualism did not need to be dwelt on, as few would maintain that the sex instinct supplied the only dynamic force to be considered in the development of the neuroses and the functional psychoses.

One will readily admit that the principle of symbolism uncritically applied opens the way to fanciful interpretations and that it has often occurred; but the fact that this has been done is no reason for doubting its value. In the study of the trends of the psychoses, one will often find that very little interpretation is necessary in order to understand the meaning of the peculiar ideas expressed, as, for instance, in schizophrenia. The Oedipus situation will frequently be found in these cases in almost undisguised form, and it is not necessary to consider the rôle of suggestion as one has to make merely a careful study of the patient's spontaneous utterances; these will often tell the story themselves.

Dr. Kirby discussed one point mentioned by Dr. Mills in his abstract which was not dwelt on particularly in his presentation, namely, that psychanalysis has proved a failure in psychiatry. From the standpoint of the results of treatment of institutional cases, the assertion may appear to have some justification. He said that few claims had been made as to the cure of constitutional psychoses or functional psychoses through psychologic analysis. On the other hand, the speaker felt that not a single public institution, and few private ones, for mental cases in this country were equipped with facilities and personnel to investigate adequately and treat patients. Nevertheless, several studies have appeared, chiefly of an experimental kind, which show that even chronic functional psychoses are often greatly improved or that even a social recovery is obtained by a careful psychologic investigation of the case, accompanied, of course, by appropriate efforts at rehabilitation.

Considered from the point of view of a method of study and investigation, Dr. Mills' statement that psychanalysis was a failure in psychiatry was to the speaker's mind entirely unjustified. There can be no denial of the fact that the psychologic investigation of the psychoses had thrown a flood of light on the development of mental symptoms in general; their origin and meaning to the patient; their significance in the general clinical picture had pointed to the forces which lie back of the development of the psychosis itself. In other words, this method of study has enabled us to see more clearly that mental disorder is best expressed in terms of a psycho-biologic reaction to life situations; has shown us that mental reactions can never be understood, even those accompanied by organic disease, by studying brain processes as such. We must know the life experiences, the conscious and unconscious conflicts, the aspirations and wishes of the individual and the reactive tendencies of the personality as a whole. The speaker said psychologic analysis contained nothing suggestive of mysticism nor was there anything unscientific about it. It was merely a way of getting at the facts which experience teaches are of fundamental importance in the understanding of abnormal mental states.

Dr. Charles Macfie Campbell of Boston said that from the point of principle he wanted to bring up the subject of the mode of presentation. Dr. Mills, with his large experience, had thought it fitting to use the tone of ridicule. He judged therefore that he considered the whole work, taught by what is widely called the freudian school, so dangerous that any weapon was justifiable. On the other hand, it seemed to Dr. Campbell that the introduction of such an attitude prejudiced the whole subject and made it extremely difficult to carry on a discussion.

Most physicians have known that considerable help can be given to nervous patients by sympathetic talks, and Darwin in his "Life and Letters" told how his father got beneath the physical complaints of women to real disturbing factors of their life.

As to the degree to which examinations are carried, in some cases no doubt after a brief discussion with the patient, difficulties could be discovered which might be easily eliminated by a frank talk. In the concrete case Dr. Campbell wondered whether the principles of the method of attack adopted by Dr. Mills were so fundamentally divergent from those of the psychanalyst, of the pupils of the freudian school. Did he not also try to understand the determinants of the symptoms presented by the patient? Dr. Campbell asked whether Dr. Mills realized that he could not find out all the relevant facts in the first interview. Dr. Campbell asked also whether Dr. Mills accepted the fact that in the determining factors of our life there are few elements more important than the early influences of the home and our relationships to our parents.

Simpler terms might be used, stating that the child has had early antagonism to the restrictions of the father; it might be said that he had an undue emotional dependence on the mother, but if another person chose to use the term "Oedipus complex," Dr. Campbell thought that he should be allowed to use his own language.

When Dr. Mills said, however, that all of the discussion of the unconscious was nothing more than a reference to what had already been presented much better under the term of heredity, Dr. Campbell wondered if that was not exaggerating the contributions which heredity had made to psychology. To his mind studies on heredity had contributed a modest amount to the knowledge of the complex determinants at work in the psychoses.

Dr. Campbell asked whether there was any psychiatrist studying intensively the deteriorating psychoses, who was not wittingly or unwittingly using the principles emphasized by Jung in his "Psychology of Dementia Praecox," based on the freudian psychology.

Some people are interested in the causes of cancer; others are interested in the treatment of that disease, and so with psychopathology and psychotherapy a person ought not to be abused because he had not made an advance in the field in which he was not working. The value of the contributions to psychology of the freudian theories is not so much along therapeutic lines, as along lines which are going to be of profound influence with regard to education.

Dr. Joseph Collins of New York stated that his qualifications for participation in the discussion were that he had carefully observed the freudian movement for twenty-five years, and for ten years had been an active practitioner of psychanalysis.

He had no intention of attempting to point out in the brief time allotted him how the freudian doctrine was rooted and nurtured in neoplatonism. Any one who wished to learn could do so readily by reading Dunlap's little book. Neither did he intend to decry the freudians. However, Dr. Collins made the dogmatic statement: the goal of psychanalysis is to expunge the moral faculty in man; its acceptation, as propounded by its founder means abandonment of the ethics and teachings of Christianity, and any one who did not see this was mentally blind or morally oblique.

To the qualifications that the psychanalyst should have, enumerated by Dr. MacCurdy—that he should have both a medical and a psychiatric education—Dr. Collins stated that he also should have an established reputation for honesty and integrity. He should neither have it in his past that he has had to flee his native country for infraction of its laws, nor should he give six weeks' courses of instruction which purport to make psychanalysts of laymen, often lewd laymen.

Dr. Collins was anxious to hear what the members of the American Neurological Association, whose observations and conclusions he had learned to trust, had to say about psychanalysis as a therapeutic agent based either on their experience or on their examinations of its doctrines with their trained minds. He knew what the professed psychanalyst thought about it, but wanted to hear the views of the man who was entitled to be heard. Dr. Collins thought it was a diagnostic or investigative measure of great value, but a therapeutic measure of mean value. It has, however, been a good thing for medicine just as every other recrudescence of neoplatonism has been. Would any one deny today that medicine has not profited by Hahnemannism, or that Christian science had not given a great boost to neurology and psychiatry? Those stately edifices are no more firmly foundationed in mysticism than the freudian teachings.

Psychanalysis as a pedagogic measure, as a method of investigation, as a penetrating illuminating trend in psychologic research is one thing: to be investigated, to be carefully considered and evaluated and to be utilized. The origin of the neuroses and the majority of the psychoses in the incest complex is another thing: to be denied, to be despised and to be treated with contempt. If the cause of the neuropsychoses is in some unadmitted sex desire, the way to cure it is to remove the cause, that is, to admit the perversion and gratify it. The freudians dare not say this now, but a few years hence they will say it unless God in his infinite wisdom will send us a new form of mysticism, and we are ready for one as heretofore the visitations have occurred in waves about thirty years apart. When it comes, our appetites for the supernatural will be temporarily satisfied by this more alluring pabulum.

Dr. Collins echoed the sentiment of those who disparaged the discussion of this topic from the point of view of levity, from the point of view of ridicule or from the point of view of pornography. He stated that for twenty-five years he had listened to Dr. Mills describe, narrate and discuss papers before the Society but he had never listened to him with greater admiration. He had never been more willing to call him master than after he had listened to his paper. He had shown a grasp of the subject from a theoretic point of view and had put his fingers on the weak spots of the doctrine and of the principles and of the argument, and in a measure of its application, as few men are capable of doing.

In conclusion Dr. Collins stated that no one could injure his amour propre by calling him a psychanalyst, but he did not think to add to his stature by calling himself one.

Dr. Adolf Meyer of Baltimore did not believe that the discussion had added materially to the possibility of clearing up the psychanalytic question—about as little as the interesting book of Mark Twain's had settled the problem of health cults, although it was an interesting and fascinating product of a man whom we honor, respect and admire very much. He did not think that Mark Twain's production went very far in making unnecessary the spreading of the Christian science movement and the so-called new thought idea. He felt much the same way about Dr. Mills' remarkable presentation.

Dr. Meyer said the only way really to approach this problem was to see that we actually turned to that with which psychanalysis dealt—a serious study of dynamic psychology, with actual work rather than production of literature. Up to this date there were in this country no organizations which had given an unequivocal practical support to honest work on dynamic psychobiology; hence, the principal exploitation came through uncritical and flashy literature and wholesale rehearsals in colleges to interest the instructor instead of getting students to do actual work. He thought that as long as the American Medical Association published editorials with so little recognition of the efforts of those who wanted to do serious work in the study of personality and life problems, as long as the financial foundations, which seemed to be the only sources from which money could be obtained for medical research, were adverse even to sincere and critical attacks on this field, just so long would there be trouble from laymen's exploitation of psychanalysis.

Dr. Mills mentioned the "Oedipus complex." Dr. Meyer said that he had a rather strong feeling against the promiscuous use of that term, but his feelings were softened rather than hardened when he saw it misinterpreted in this sense. Freud found himself stirred to describe a life situation in that way, a life situation which makes an impression on people because it touches certain tendencies and fears which no other description would adequately describe. He used the Oedipus myth and its meaning to the Greek world as an illustration of the potency of incest fears. Dr. Meyer said that this was true of many psychanalytic terms. Expressions are used which are perhaps bungling and open to misinterpretations, but at least they bring out new facts as to the native tendencies of man. We could not reduce all these concepts to the principles of reflex-physiology of a necessarily narrow and static type. Neither the American Neurological Association nor any other association should close the door to honest attempts to find new terms for new facts. The chief aim should be to see that there were means furnished to study those facts, and that there should be more work, rather than literature and scintilating talk.

Dr. Charles K. Mills,[1] of Philadelphia, in replying to those who discussed and criticized his paper, said that it first must be emphasized that in a presentation of a subject like psychanalysis to a medical association, time did not permit of its thorough and elaborate consideration.

It has been asserted that neurologists, on the one hand, and academic psychologists, on the other, are not fitted to pass judgment on psychanalysis, but if those who are in favor of psychanalysis and who practice it professionally are the only ones who have a right to speak on the subject, its discussion would be altogether one sided and would not tend to the advancement of the truth. A correct judgment may be reached by a careful study of the literature of psychanalysis, by a consideration of what appears to be its fundamental principles and its methods of practice, and by observation of its effects as observed in the profession and the community.

With regard to Dr. Mills' personal experience, he said that the forms of nervous and mental diseases which have received most attention from the psychanalysts have been for many years under his observation and care both in private and hospital practice. All successful neurologists and alienists have been in a broad sense psychanalysts: Charcot, Gowers, Mitchell, Seguin, Janet, Prince and many others who might readily be named have been among those who successfully practiced psychanalysis and psychotherapy in their own way. The results obtained by these men were as good as those which are claimed by the psychanalysts of the present day.

A proper attention to sexual etiology was not overlooked by the men of this class. Every neuropsychiatrist has had much to do with certain varieties of sexual neurasthenia, or sexual anxiety neuroses, the two designations covering much the same class of cases although a separation between them might in some instances be made. In former days as in the present, many of the cases chiefly exploited in freudian literature were improved or were cured, in so far as such cure was possible, by well known physical and nonfreudian methods.

Dr. Mills went early into the field of hypnotism stimulated largely by a knowledge of the work done by Charcot, and Richer at the Salpétrière, and his experiences in this field should he believed entitle his views to some consideration. Hypnotism and psychanalysis have not a little in common, a fact which has been clearly recognized by such analysts as Ferenczi and even by Freud himself, although the latter discarded hypnotic procedures entirely as he advanced in his study of psychanalysis.

Dr. Prince, Dr. MacCurdy and Dr. Macfie Campbell lay stress—too much stress Dr. Mills thought—on what they asserted was his too great use of ridicule in attacking freudian psychanalysis. He would simply say that it was not his intention to resort largely to this weapon, sometimes allowable, in pronouncing his views on the subject. It was not to be put entirely to Dr. Mills' account if some of his auditors in some places chose to see a humorous side to the subject, or if you chose, of his manner of presenting it. If not to freudian psychiatrists, at least to many thinking men, some of the interpretations of dreams and direct analysis to be found in psychanalytic literature could not but make the recorders of the data appear to be too credulous and lacking in scientific acumen.

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[1] At the meeting of the Neurological Association at Atlantic City the time was exhausted before Dr. Mills had the opportunity of replying to those who discussed his paper. The manuscript of these members of the Society has been submitted to him and his remarks in closing the subject are in the main based on this manuscript.

Is it to be held against him if others like himself found something foolish or absurd in the alleged symbolism of the "tunnel window" incident? Could he be seriously criticized because of his allusion to Michael Angelo and others who have been, as he believed, misrepresented by legends to their artistic productions? The manner in which Dr. Mills had spoken of ancient sophists comparing their terminology with that of modern freudian sophists was not, he believed, unjustifiable.

Dr. Mills said that the object of his paper was not simply to ridicule psychanalysis, but rather to give in a series of briefly presented propositions his main reasons for disbelief in recent psychanalysis and especially freudianism. It might be well for him to recall categorically although briefly these propositions. In the first place, he spoke of the tendency to mysticism or semimysticism as evidenced by different psychanalysts and, curiously enough, as regarded by different members of the psychanalytic school in their comments on each other. He also spoke of their undue use of terminology, a subject which has attracted the attention of others both abroad and in this country. The differences among psychanalysts in their interpretation of the unconscious, their incorrect methods of making use of dream analysis, and their unwarranted employment of sexual or phallic symbolisms in both dream and direct analysis were emphasized by him. In pointing out the possible benefits to be derived from psychanalysis, Dr. Mills indicated, although very briefly, the fact that freudian psychanalytic literature had done much to turn the attention of the profession to the importance of the sexual factors in the investigation of psychoneurotics. In speaking of the evils of psychanalysis, Dr. Mills called attention to the general influence of the propaganda on the moral tone or fiber of the profession and the community, and he also spoke of the particular evils which might result from the so-called process of transference, a point to which his critics had given but little attention. With regard to the subject of psychanalysis and the experiences of the recent great war, he was largely in accord with his critics and others although he did not think that psychanalytic writers had given sufficient consideration to the question of the influence of organic factors in the production of war neuroses and psychoses.

The word "dynamic" had been used in the discussion and in psychanalytic literature in a sense which was at times misleading. Every neuropsychiatrist recognizes a dynamic psychology and a dynamic pathology, but the psychanalysts of various schools at times use this term in a way that tends to the inference that there is a dynamism which has no structural machinery back of it. Dr. Prince, in private conversations and to some extent in public discussion, has shown what seems to Dr. Mills to be a decided tendency to disregard organic neurology, as for instance in his impatience to give it any consideration when describing such subjects as hallucinations.

Dr. Prince accords high praise to Freud for the doctrine of repression and conflict, but, after all, is this doctrine of repression and conflict—of course, not now using these terms simply in a freudian or sexual sense—any more due to Freud than is the doctrine of the unconscious which Prince properly refers to a group of philosophers some of whom long preceded Freud? The idea of repression and conflict permeates psychologic medicine and is exemplified in much that has been written about such a simple subject as worry, which for generations has been so frequently presented as an important etiologic factor in psychoses and psychoneuroses.

With the remarks of Dr. Schwab and Dr. McCarthy Dr. Mills was largely in accord. What had been said by Dr. Schwab regarding his experiences in France during the war coincided with the views of Dr. Mills as expressed in the body of his paper. Dr. McCarthy was a little more generous than Dr. Schwab to recent psychanalysis in the enumeration of his war experiences. Here and there Dr. McCarthy's remarks were somewhat delphic, not clearly favoring either the psychanalysts or their opponents.

Both Dr. McCarthy and Dr. Schwab indicated that in their war psychotherapy they used the older so-called "Mitchell method" quite as much as those of psychanalysis.

With regard to the remarks of Dr. MacCurdy, Dr. Mills had little to say in disagreement. Dr. MacCurdy seemed to have recognized what Dr. Mills believed he had recognized, namely, that the sexual aspects of psychanalysis had proved a dangerous lure to many in the community. Dr. Mills believed that three requirements given by Dr. MacCurdy for successful practice of psychanalysis were those which would be requisite for any method of treatment requiring intelligence on the part of the patient.

As to psychanalysis passing into the discard, Dr. Mills thought that this was true with regard to freudian psychanalysis although it was quite possible that it might to some extent tend to recrudesce as spiritualism, as all other false medical cults had a tendency at times to show their heads again.

With the remarks of Dr. Kirby, Dr. Mills said he would considerably but by no means fully agree. Dr. Kirby intimated that the freudian standpoint and the theory of pansexualism had been largely abandoned. While this might be true, Dr. Mills felt that so far as his study even of the recent literature of the subject had gone, sexual theories, largely freudian, still held a prominent place in psychanalysis. Dr. Mills said he did not attend the recent meeting of the American Psycho-Pathological Association, but was glad to hear from the speaker, and also from Dr. Prince and others that the freudian shackles were being loosened.

With regard to the psychobiologic approach to questions like the nature of the psychoneuroses and neuroses, Dr. Mills said he was certainly in accord as to its value, but simply wished to insist that this psychobiologic approach should not be restricted too much to purely sexual channels.

Time had not permitted him to discuss the question of insanity in its relation to psychanalysis. He believed, however, that the general statement of the preliminary analysis was correct, namely, that psychanalysis had not proved a success in the therapy of mental cases.

Dr. Mills said that he disagreed with Dr. Kirby when the latter said that there was nothing of the mystic element in psychanalysis. Certainly there should be nothing of this kind in genuine healthful psychanalysis, but Dr. Mills believed that he had made clear his point that any thoughtful student of the well-known literature of psychanalysis could not help see in it a tendency to mysticism and he would like again to call attention to what he referred to in the paper as presented, namely, that some of the strongest protagonists of psychanalysis had discovered or believed they had discovered a mystic or semimystic element in the work of their confreres of some other subdivision of the general psychanalytic school.

With reference to the remarks of Dr. C. Macfie Campbell, Dr. Mills said that he undoubtedly in his practice tried to discover the determinists of the symptoms which were presented by his patient. Every practitioner of neuro-psychiatry of any force or standing does this, but he does not and should not lay all or even the greatest stress on the sexual history of his patient unless it became clear in his investigations that it was this which played the chief rôle. As in the reference made by Dr. Campbell to the father of Darwin, it is certainly desirable in studying the affections of nervous women and everybody else to get a sympathetic insight into the causes of their nervousness, but does this mean freudian psychanalysis? One of Dr. Mills' objections was to the prolonged probing to which patients were often subjected, if he was to judge from the literature of psychanalysis and from conversations both with psychanalysts and their patients. He hardly thought it necessary to take weeks, months and even years to get a sympathetic insight into the nature of a case. He was sure in some instances of which he had knowledge that this prolonged probing had done more harm than good. Dr. Mills thought that Dr. Campbell had misunderstood his allusions to heredity. It was in connection with the views of Jung as emphasized by Nicoll that he had spoken of this subject. These gentlemen seemed to build up out of the unconscious of many generations a pet theory of the unconscious. One does not inherit the unconscious, what one does inherit is traits, trends and tendencies.

Dr. Mills agreed largely with all that Dr. Collins had said except of course his too complimentary remarks about himself. He thought he was right about neoplatonism, which was in a way a corroboration of the views expressed in his paper. He believed also that the too ardent pursuit of sexual psychanalysis certainly tended to undermine ethics and perhaps Christianity.

If Dr. Meyer in his reference to Mark Twain had Dr. Mills in mind he would only say that he thought that Dr. Meyer had been a little hard on this distinguished humorist with whom Dr. Mills said he had no right to be classed.

THE ANATOMIC SEAT OF THE EMOTIONS: A DISCUSSION OF THE JAMES-LANGE THEORY*

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It is now thirty years since Prof. William James and Professor Lange independently stated their theory of the mechanism of the emotions. This theory has never been satisfactorily proved or convincingly refuted.

JAMES-LANGE THEORY OF EMOTION

The theory, as stated by James is, briefly, that the mental state experienced in emotions of fright, anger, joy, etc., is due to different sensory impulses sent up from the muscles, viscera, blood vessels and the periphery in general. It is the palpitations, tremors, abdominal qualms and other peripheral disturbances which cause the emotion. "One is afraid because he runs; he doesn't run because he is afraid." "One is pleasant because he looks pleasant." These are rather exaggerated illustrations. Attempts have been made to strengthen this phase of the theory by obtaining the opinions of great actors as to their real emotional state when they were expressing emotion. The inquiry did not help the theory, for the experiences were conflicting.

It may be interesting to return to the neurologic quest for which James first appealed, and to see whether later experience throws any light on the subject.

Professor James thought that if he could find a case in which a person was anesthetic both without and within, and if such a person felt no emotion, it would support his theory. He did find three cases of this kind, but they were not adequately reported and not considered evidential. The subject appears never to have interested neurologists very much. Of late, however, such interest has been aroused because of the emphasis that is being placed on emotion as a pathogenic agent, and also as a kind of measure of cultural progress, for it has been noted that where emotion dominates, civilization halts or disappears. With this increased attention to emotional factors, there has come a leaning to the view that the James-Lange theory or some modification of it is true. Some seem to think that it is the perturbations of function in the splanchnic or vegetative nervous system that especially arouse the mental experiences in emotion. Others suggest that in addition the endocrine glands and the hormones are important peripheral agents in arousing emotional states.

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* Read at the Forty-Fourth Annual Meeting of the American Neurological Association, Atlantic City, N. J., June, 1921.

On the other hand, Sherington reports one experiment, on a dog, in which he severed the cervical cord and the vagi, and found that he could still arouse evidence of emotion in the animal. This, however, is only one case and is not convincing.

The possibility of finding such cases of anesthesia as James was looking for is very remote. They could only be aroused by an extensive double lesion of the thalami. I have seen a case of complete one-sided anesthesia due to such a lesion, but with no special emotional disturbance. Sometimes a thalamic lesion causes paralysis of emotional facial expression, without particularly disturbing the emotions. One can fairly assume that if there were a double thalamic lesion causing such paralysis, the patient would feel the emotion associated with laughter or tears, without any peripheral muscular movements, and consequently no afferent reaction from the somatic system, at least. Such a patient would feel joy or fright, but could not express it in any of the ordinary muscular movements. In so far as they go, these facts tend to disprove the James-Lange theory.

If an object suddenly arouses an emotion of fear, for example, efferent impulses must be sent to the periphery before afferent impulses can be sent back. In neurologic experience we do see cases in which this efferent part of the reflex process and sometimes both pathways are very largely blocked. It is cases of this kind that I wish to report and to interpret.

Before going further let me state what meaning is attached here to the technical terms used in discussing our subject.[1] The characteristics of emotion, as described by James, in 1890, fit very well into the description of today. We say, as James virtually said, that an "affect" of "feeling tone" is something connected with sensation and has a peripheral origin. The appetites, desires, the sense of ill-being or well-being, hunger and fatigue are associated with one or more of our now estimated twenty-three special and general sensations. They are "feelings" or "affects."

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[1] The brain-stem to which I refer later is generally described (Villiger) as including the medulla, pons and corpora quadrigemina. Tilney in his "Form and Function of the Nervous System" includes the optic thalamus. There is a difference among writers in the terms used in describing the sympathetic nervous system. The more common nomenclature is that which calls the "sympathetic nervous system" that part which is connected with the dorsolumbar portion of the spinal cord. The autonomic or "parasympathetic nervous system" is that which comes from the midbrain, medulla (vagus) and sacral part of the spinal cord.

An emotion, however, is always connected with a percept, idea or concept which arouses one of our primitive instincts, these being mainly variants of fear or love.[2] There is, said James, always a bodily sensation with emotion and generally some motor bodily reaction which has about the same pattern for the different emotions; that is, all persons react in about the same way motorially to fear or to joy or to anger. James emphasizes the fact that with emotion, at least with all the primal emotions, a person has some bodily sensation, such as a thrill, tremor, palpitations, abdominal qualm, faintness, etc.

EFFECT OF SOMATIC OR SKELETAL MUSCLES AND SYMPATHETIC SYSTEM ON EMOTIONAL STATES

Returning now to the James theory, I wish to present certain clinical facts, which seem to me to show that the somatic or skeletal muscles and sympathetic system proper have at least nothing but a minor and contributing effect in arousing the conscious emotional states.

I had a patient, a very intelligent woman of 40, who fell from a horse and broke her neck at the third and fourth cervical level. She was completely quadraplegic and suffered complete loss of cutaneous and deep sensation from the neck down—with abolition of all deep reflexes. A roentgenogram showed the presence of the fracture.

She lived for nearly a year, and during that time I saw her showing emotions of grief, joy, displeasure and affection. There was no change in her personality or character. The only skeletal muscles at her command were the cranial, the upper cervical and the diaphragm. As for the vegetative nervous system, she had the vagus, but the vagus has no active (low-level) sensory fibers except the laryngeal. The parasympathetic system in this patient was able to function, but the sympathetic proper was absolutely eliminated from subconsciousness and consciousness. It is difficult to understand, on the peripheral theory, why there should have been no change in her emotionally, with the skeletal system practically eliminated and the sympathetic entirely so.

Such a case as this is not so uncommon; I have seen others with similar symptoms, and they are very familiar to neurologists, but the reported case was unique in that the patient lived so long in such a normal mental condition.

Turning to other neurologic fields, we have all seen patients in the terminal stage of tabes, with perfectly atonic, anesthesic and greatly wasted skeletal muscles. In my experience their emotional reactions were present and not abnormal.

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[2] McDougal enumerates six instincts with associated emotional states.

I had once in the hospital a patient with family periodic paralysis, who for a few days was completely paralyzed except for the muscles of mastication, deglutition and respiration, but with only the natural emotional and mental changes.

In the finally complete bodily immobility and paralysis of certain forms of progressive muscular atrophy, in the absolute bodily rigidity of terminal arthritis deformans, and in the advanced stages of paralysis agitans with rigidity, all muscular movements are practically eliminated, except those moving automatically and those under the influence of the vegetative nervous system. It is at least my experience that in these patients the emotional reactions are present and normal to their condition. In all these cases the vagus system is able to act, but not the somatic, and in some cases not the sympathetic.

It does not always follow that because a sensation is felt in the periphery it is due to peripheral irritation. We can have pains in the face or arm from lesions of the thalamus, and we can have thalamic paresthesia and dysesthesia. This is shown in lethargic encephalitis and in thalamic and midbrain diseases. The dysesthesias of Parkinson's disease may be in part cerebral. In fine, we know that some of the bodily sensations, such as are sometimes associated with emotion, especially anxiety states, may be central and thalamic; so that the fact that with emotion bodily sensations are felt does not prove that they come from the periphery.

Expressions of emotion, in the form of tears and laughter, occur automatically in lesions of the brain-stem and thalamus. These outbursts are not usually associated with any real emotion of sorrow or joy; and an argument based on these cases can be made both for and against the view of a peripheral source of emotion. Thus, if the source of the emotion is peripheral, why does not the man with a thalamic lesion who bursts into laughter or tears, have eventually a real emotion of joy or sorrow, since all the somatic and splanchnic changes that accompany emotion are present? On the other hand, if emotion is of central origin, why does not the person with a thalamic lesion have emotional states as a result? I would answer in the latter case that it is because in thalamic lesions there is a rupture of the relations between the thalamus and the cortex, for there must be a corticothalamic and thalamocortical activity in the emotional experiences.

A person may have a palpitation of the heart or a sense of faintness or tremor or sweating without experiencing any emotion of fear or apprehension, unless finally the idea that he is in physical danger comes to the mind. This fact is explained by various psychologic mechanisms. It seems most reasonable to suppose that when a palpitation is felt the emotion of fear develops, because an association process arouses an idea of illness, and this causes an emotion of fear. The mechanism of an "associated conditional reflex" may also be effectively invoked in explanation.

Finally, neurologic experience also throws doubt on a certain psychologic view, lying close to the James-Lange theory, to the effect that thought and emotion are always at once reflected out upon the muscles which in turn react and return afferent impulses to the brain. The statement is made that "we think with the muscles" because every thought is accompanied by muscular movement and every muscular movement arouses some "feeling." The muscles of the larynx and articulation are assumed to do this in particular. Even in abstract thought or meditation some slight muscular action is assumed to occur in the fine muscles of the larynx.

The neurologic experience is, however, that a person continues to feel and think perfectly well after the larynx has been removed or paralyzed and also when there is a complete muscular paralysis.

AUTHOR'S THEORY OF EMOTION

On the whole, I am led to the conclusion that emotion is centrally located and results from the action and interaction of the cortex and thalamus. The bodily sensations which accompany emotion are produced by stimuli from the automatic centers in the brain-stem (acting on heart, blood vessels and glands), but they only cooperate to extend and perhaps intensify the emotion. For naturally, if a man finds his heart palpitating, his hands cold and wet, his legs weak and his epigastrium calling out in distress, he will feel more alarm. On the other hand, if the automatic stimulus produced by joy at the sight of the beloved, strengthens his heart beat, raises his blood pressure and in general improves his sense of well-being, he will feel a little happier for it.

The centers that control the activities of the vegetative system are mainly in the brain-stem.[3] These centers are first appealed to when the animal perceives something calling for instant action in defense, offense or active desire. They, on the other hand, stir up the muscles, viscera and glands, but they also appeal through the thalamus to the cortex and arouse the emotions appropriate to the object recognized or idea that has been aroused. As the suprasegmented nervous system develops, emotion develops.

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[3] In a recent address before the New York Neurological Society Prof. Walter B. Cannon presented evidence to show that the center for sympathetic discharges occurring on pain-stimulation lies in the brain-stem as high up as the anterior corpora quadrigemina.

I am inclined to the opinion, then, that the James-Lange theory of emotion is true only in part; that is, that the peripheral visceral stimuli are only later and contributing factors to emotion, and that the skeletal muscles and sympathetic system do not play an essential part.

I am not presenting all the data possible bearing on this question. The experiments and operations on the sympathetic ganglions, for example, show that the cerebrum can get along quite well without them or without there being much connection between these ganglions and the central nervous system. Arguments against the peripheral theory also have been presented based on studies and experiments with the glands of internal secretism. I do not attempt to introduce these data which will be found in the literature of endocrinology.

My object has been to present some facts with deductions therefrom and to elicit further contributions from the experience of neurologists.

FRONTAL LOBE PHENOMENA

AS OBSERVED IN A CYST IN THE LEFT FRONTAL LOBE; MARKED REMISSION OF SYMPTOMS; DECOMPRESSION AFTER THIRTEEN MONTHS; NECROPSY

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The clinical phenomena associated with lesions of the frontal lobes of the brain have shown a greater diversity in character and degree than those of any other locality. Not infrequently, they have been so negligible as to have left this region unsuspected during life. With more or less hesitation, neurologic clinicians have ventured syndromes as characteristic which have varied between wide limits.

Tumors of the frontal lobe have nevertheless been encountered quite frequently, as may be inferred from the series collected by Clarke,[1] in which there were fourteen instances in ninety-six cases of brain tumor.

Oppenheim [2] has said, "Apart from aphasia we know of no characteristic localizing symptom of tumor of the frontal lobe."

The difficulty of localizing by clinical signs is unusually but significantly expressed by T. Grainger Stewart [3] when he says, "The clinical picture of frontal tumor is characterized by *the absence* of signs pointing to involvement of other regions of the brain rather than by the presence of special focal symptoms." According to the same author, the general symptoms are usually present, namely, headache, optic neuritis and vomiting.

As a result of their experience with war injuries, Marie and Béhague [4] have concluded, after many observations, that deep lesions of the prefrontal region give rise to a syndrome characterized by disturbance in orientation in space, while there is an absence of all objective signs of a lesion of the nervous or vestibular systems, and headache, vertigo and dizziness were entirely absent. In the course of the same report, however, it is admitted that "in spite of controversy, the question of the physiological rôle of the anterior part of the frontal lobe remains obscure." Chatelain is quoted: "Up to the present time there is no true symptomatology of the frontal lobe."

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[1] Clarke, Frederick B.: A Study of the Anatomical Location and Histopathology of Ninety-Nine Brain Tumors, Rev. Neurol. & Psychiat. **14**: No. 11; abstr. J. Nerv. & Ment. Dis. **48**:498-500, 1918.

[2] Oppenheim: Textbook of Nervous Diseases, Trans. by A. Bruce, 1911, p. 900.

[3] Stewart, T. G.: The Diagnosis and Localization of Tumors of the Frontal Regions of the Brain, Lancet **2**:1209-1211, 1906.

[4] Marie, Pierre et Béhague, P.: Sydrome de désorientation dans l'espace, consécutif aux plaies profondes du lobe frontal, Rev. Neurol. **35**:3-14, 1919.

Among the symptoms mentioned during a recent discussion of the subject in Leipsic, [5] ataxia of the trunk, akinesia, drowsiness, lessened activity, rigidity and fixed positions, were especially emphasized.

The frequency of muscular movements resembling those of paralysis agitans was brought out—the general impression being that these as well as ataxic symptoms were to be attributed to implication of the fronto-cerebello-pontile tracts. Likewise, symptoms referable to the posterior fossa have been frequently observed, in many of which the frontal lobe was not suspected until the necropsy.[6]

Similarly, one of the patients in the three cases of Dercum [7] was operated on subtentorially, because of cerebello-pontile symptoms, but no tumor was found; the postmortem examination revealed a tumor in the left prefrontal region. A second patient presented the characteristic described by Marie [4] in that he became lost in his own house; a tumor in the right frontal lobe was found at necropsy. The third tumor was correctly localized and removed from the right frontal lobe. In two of the cases an optic neuritis was present.

Epileptic seizures were frequently observed in nineteen of twenty-two of Stewart's series, who also quotes Müller as finding convulsions in one-third of his 168 cases. Such attacks have generally been regarded as indication of pressure on the anterior central convolution posteriorly. However, as was the experience of Foerster,[8] it was only by the revelation of the roentgenogram in a case with convulsions that the frontal lobe was incriminated and a psammoma successfully removed.

McConnell's [9] patient who had a tumor involving the first and second convolutions of the frontal lobe presented disturbance in writing when he made a voluntary attempt or when he wrote spontaneously, as well as when he wrote from dictation, while when copying his writing was "practically perfect." His speech was thick which "appeared to be due to paresis of the muscles of articulation, enunciation and phonation." Mention is made of omissions, faulty spelling and formation of words in a letter written two years before observation. Memory and attention had failed. There were convulsions and hemiparesis of the face.

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[5] Jahres Versamml. d. ges. Nervenärzte, Neurol. Centralbl. **19**:649, 1920.

[6] Ruckert, A.: Ein Stirnhirntumor unter den klinischen Bild eines Tumors der hinteren Schädelgrube, Berl. klin. Wchnschr. **46**:1248-1251, 1919. Souques, A.: Des Troubles Auditifs dans les Tumeurs Cerebrales, Rev. Neurol. **12**:727-731, 1904.

[7] Dercum, F. X.: A Report of Three Prefrontal Tumors, J. Nerv. & Ment. Dis. **37**:465-480, 1910.

[8] Foerster, O.: Discussion: Zur Diagnose von Stirnhirntumoren (Bostroem, A.), Neurol. Centralbl. **19**:650, 1920.

[9] McConnell, J. W.: A Case of Tumor of the Left First and Second Frontal Convolutions with Motor Agraphia as Its Chief Localizing Symptoms; Successful Removal of the Tumor, Univ. of Pa. Med. Bull. **18**:1905; abstr. J. Nerv. & Ment. Dis. **33**:211, 1906.

A tumor was removed by operation which was lying across the foot of the second frontal convolution, encroaching on the lower half of the first, slightly on the posterior portion of the third and the anterior edge of the precentral convolutions.

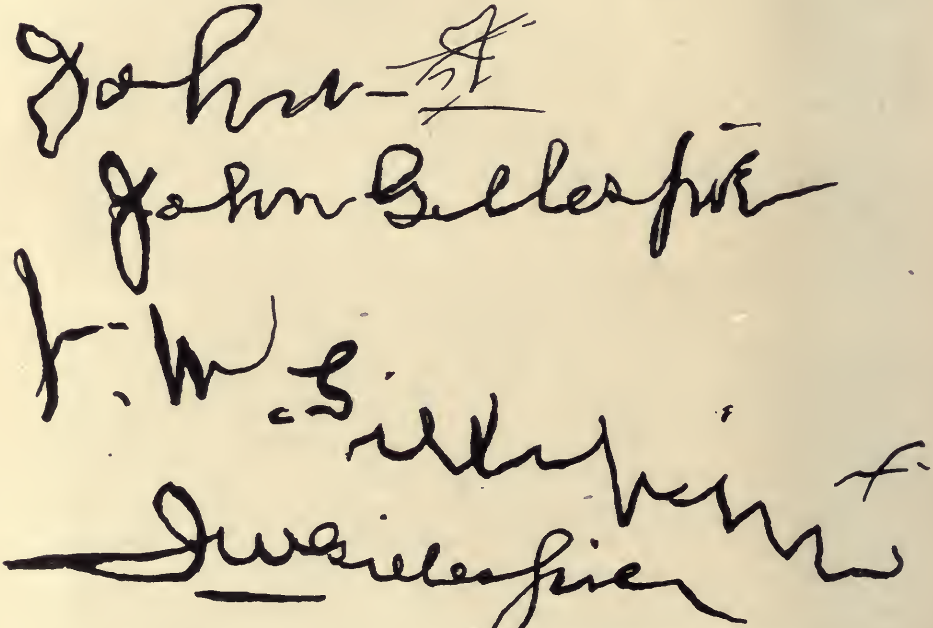

Fig. 1.—The various changes in handwriting as improvement progressed.

Unilateral anosmia and the occurrence of optic atrophy on one side with more or less neuritis on the other have been cited, but obviously could only apply where the location closely approximated the inferior surface of the frontal lobe.

Psychic symptoms are not infrequent, according to Stewart, but are probably greatly exaggerated. These concern chiefly disturbance in attention, delayed response or other evidence of slowness in thinking; various degrees of loss of memory; some irrelevancy with jocularity; apraxia in general and as related to speech; the disturbance in orientation, especially in direction or space, rather than with reference to time (Marie);[4] and as regards cerebral activity, from all degrees of coma and stupor to mania with active delusions.

In arriving at a diagnosis of frontal lobe tumor, Grainger Stewart[8] says that we have to rely on (*a*) the presence of the general symptoms of an intracranial growth, (*b*) the presence of mental symptoms, and (*c*) the absence of focal signs pertaining to other regions of the brain.

The case which I report presents not only a verified lesion of the frontal lobe without general symptoms and with an absence of the frontal lobe symptoms mentioned, but also includes speech and graphic disturbances with a predominance of apraxia as related to these functions as well as to movements with less highly developed purpose; and these were sufficiently distinct to direct the attention to the left inferior frontal lobe. These manifestations and the intellectual state are more interesting because the patient was a practicing attorney of prominence and reputation, whose preliminary training included a university course in arts, followed by a baccalaureate in law from the University of Michigan.

REPORT OF A CASE

History.—J. W. G., aged 44 years, unmarried, attorney at law, was first observed Feb. 17, 1919, complaining of vertigo and slight edema of the hands which had been noticed during the past two or three years. His mother had died suddenly a year before (apoplexy). His father was in good health. He had no brothers or sisters. A paternal grandfather died of cancer. Otherwise his history was negative as to constitutional, nervous or mental disease. He had had measles, whooping cough and mumps in childhood. No illness other than attacks of biliousness were admitted. Gonorrhea and syphilitic infection and its manifestations were denied; there was no alcoholic excess. There had been no serious injury and he had never been unconscious from any cause. He had always been robust and athletic in development and practice.

The present complaints included vertigo and transitory edema of the hands. There was no headache, no vomiting or numbness. A zig-zag scotoma was admitted. Dizziness was increased on stooping and was characterized as "bilious." His appetite was good; he had no digestive distress. The bowels were usually regular. Sleep was good and restful.

Examination.—The patient was a man of excellent development, 5 ft. 9 in. in height, weighing 178 pounds. The heart borders and sounds were normal. Blood pressure (Faught) was: systolic 140, diastolic 85. There was no motor disturbance—voluntary, associated or reflex. All deep reflexes were present, equal and about normal in degree. Ocular movements were present in all directions without nystagmus. The pupils were equal and responsive. The fundi were normal. The urine was acid; specific gravity, 1.024; no albumin; no sugar; no bile; no casts—cylindroids, no epithelium.

Second Admission.—July 5, 1919, the patient returned complaining of numbness of the right hand—more marked in the thumb and index fingers. Difficulty had been noticed in the finer purposeful movements of the fingers, especially evident in buttoning and unbuttoning his coat. The sense of numbness included at times the whole hand and, to some extent, the arm and varied in degree and extent.

Second Examination.—Examination at this time failed to reveal disturbance in the detail movements of the fingers, and the associated movements were well executed. The tongue was protruded in the middle line, but heavily coated. The mouth was more movable on the left side than on the right.

The tissues of the right hand were softer and more flabby to palpation in contrast to the opposite side. Power in the right hand was slightly less than in the left; the dynamometer (Tiemann) registered: right, 140; left, 190. The reflexes of the right forearm were prompt. Faradic responses of the muscles of the right forearm were prompt, with a current of moderate strength. The pulse was slow, regular and of good volume. The optic fundi showed no change from previous observation. The urine was normal, except for a distinct trace of indican.

July 11, 1919: There was some relief following thorough eliminative medication, but attacks of vertigo were noticed and, on one occasion, he fell.

A disturbance in speech was experienced on two occasions—on one a difficulty of articulation and thickness of speech were recognized, and on another the inability to recall words in order to express an opinion during a conference or consultation in which complex legal points were involved.

The numbness directed to the thumb and forefinger of the right hand persisted. Examination at this time revealed no objective evidence. The brow was evenly wrinkled. The tongue was mesially protruded and was moderately coated. The mouth was retracted quite evenly. Associated and detail movements of the hands were well performed. The dynamometer registered: right, 290; left, 280. He walked well forward and backward with the eyes open or closed and maintained himself without unsteadiness under the same conditions. All of the deep reflexes were present and equal. The plantar reflexes were present and flexor in type.

Special Senses: Notwithstanding the subjective complaint of numbness, no disturbance in sense perception could be made out, especially in the radial area of the back of the right hand, which was felt as "numb and puffy." There had been and was some vertigo entirely subjective in type, that is, he felt as though he were turning in space.

Eyes: All external ocular muscular movement was present without nystagmus. The pupils were equal and responsive to light and distance. The fields of vision, as estimated by testing with colored pencils, showed slight contraction in the right, both as to form and color; accurate testing was not done at this time. The right optic fundus was congested and there was slight blurring of the margin of the nerve head; some tortuosity of the vessels was evident; the left fundus was normal, except for slight capillary injection.

Hearing, Taste and Smell: Hearing was not impaired as far as the speaking voice was concerned. Taste and smell were not impaired.

Course.—July 15, 1919: The patient had been recumbent for the greater part of the time and under iodin medication. There had been transitory attacks of paraphasia in which it was difficult to find the word desired, and at times words were misused. There was slight weakness of the right side of the face. Association as well as detail movements were well accomplished. Gait and station were not impaired. Ocular movements were not altered, and the pupils were equal and responsive.

July 16, 1919: After having been up, dressed and about the house, contrary to instructions, the patient became suddenly unable to speak in that he was unable to find words with which to express himself, and the attempt to do so led to confusion, perplexity and gross misplacement of words. The ability to execute muscular movements as purposed failed, whether by verbal direction or by imitation. Although able to walk, there was slight unsteadiness. Detail movements of the fingers—for example approximation of the thumb with other fingers—were lost and if exemplified could not be imitated. Instead of approximating the fingers by imitation he clenched the fist; the error was not recognized.

The facial movements showed weakness on the right side without twitching and more pronounced on emotion. There was no disturbance in coordination of automatic movements, as in walking and movements of the hands; but the acts proceeding from voluntary purpose, while coordinated, were quite foreign to purpose.

Laboratory Findings: (July 18, 1919, Dr. Ward Burdick). Blood Serum: The Wassermann test revealed 100 per cent. hemolysis with two cholesterinized and three plain antigens. Cerebrospinal Fluid: Pressure was moderately increased; the fluid was clear, transparent and limpid; it contained 10 cells; colloidal gold curve, 0000011000; globulin, present (+); Wassermann reaction, 100 per cent. hemolysis with cholesterinized antigen and 2 c.c. of fluid. Morphologic Analysis of Blood: leukocytes, 10,800 (polymorphonuclear neutrophils, 75; lymphocytes, 15; large mononuclears, 9; eosinophils, 1).

July 30, 1919: Improvement, since last notation, had been chiefly in the direction of voluntary speech. The patient was able to recognize objects and their uses readily, was able to recall objects when named and to point them out at will. Considerable difficulty was encountered in computations in figures. Oral spelling of words was difficult, and frequent errors were made in small, simple words. He was able to read understandingly and to think in speech. Musical airs were remembered when mentioned and recognized when heard. Test words were repeated with fairly good articulation. In voluntary speech, however, considerable dysarthria was manifested at times over simple words; this was especially noticeable with the contained consonants, and he would frequently repeat several words rhyming with the one he desired to use, stopping promptly when the correct word was enunciated. For example, in attempting to use the word "drop," he repeated "crock," "crop," "trop," and finally stopped with the correct word "drop," recognizing it as the one desired.

All movements were well executed, except that the emotional movements of the mouth were still more pronounced on the left side. The pupils remained small, but the vision of the right eye, especially with reference to the right field, was complained of as "hazy." The optic fundi showed no greater change than slight capillary injection without interference with the nerve margin. Numbness was persistently experienced in the right hand, more especially in the radial or area of median supply. The power of the right hand was diminished; the dynamometer registered: right, 220; left, 275. There was no change in the reflex responses; the plantar and Oppenheim reflexes were frankly flexor in type.

The fields of vision, as taken by perimeter, showed slight constriction, more marked on the right; no alteration in color relations and no interlacing was observed (see chart).

The speech disturbances were carefully studied with the assistance of Dr. C. S. Bluemel, who kindly made the following observations and notes: Aug. 6, 1919: The patient greets the doctors with some pleasure and animation and opening the door for them to enter the house, he says, "Sit down;" at the same time he nods at the chairs on the front porch with a little confusion, giving the impression that with fluent expression at his command he would have asked whether the doctors would have preferred to remain outside or go in the house.

When introduced to Dr. B. he does not readily find the conventional expressions, and he expresses the amenities more by the hand-shake, nodding and by facial expression. He shows similar paucity of verbal thought throughout the examination, and his facial expression and manner suggest that he would frequently express ideas if the words were forthcoming.

His verbal expressions are usually accurate in pronunciation, but they lack continuity or coherence, and thus his sentences or phrases seem to be hyphenated. He never indulges in any lengthy verbal expressions, but rather in short and isolated sentences. Occasionally his words seem apposite, being rather inappropriate synonyms for the words he cannot recall. Sometimes his sentences are unfinished owing to his inability to recall appropriate words.

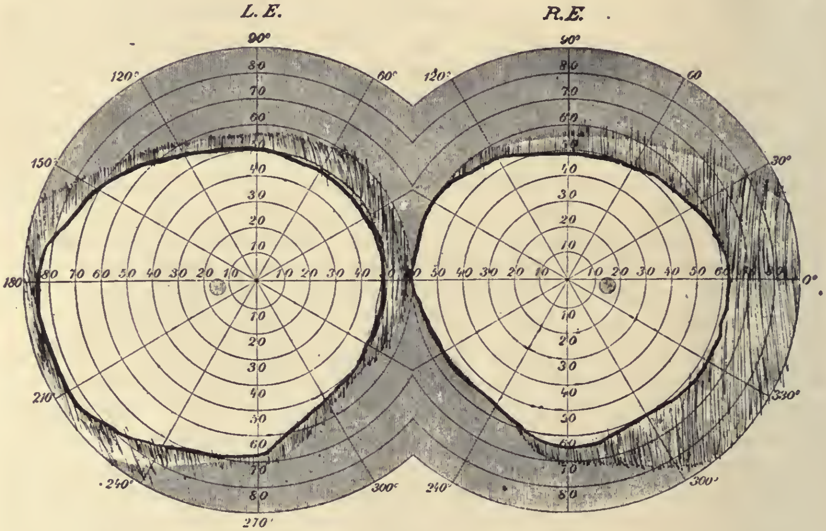

Fig. 2.—Concentric constriction of the visual fields with suggestion of right homonymous hemianopsia, Oct. 4, 1919.

His manner clearly shows that he realizes his disability and he recognizes the correct word when he finally pronounces it, or when it is supplied by another person. His chief difficulty seems to be his inability to recall the appropriate words, and he remains silent during the greater part of the conversation despite the fact that his attention is keen and his manner alert. He frequently slurs the pronunciation of words, or elides consonants or syllables. Occasionally he pronounces the first syllables correctly, then flounders with the remainder of the word or mispronounces it (dict-ionary, motor-neer for motorman).

He cannot readily recall and recite short rhymes that have been taught him. He does not do mental arithmetic well. The product of three times forty-six is given as one hundred thirty-one; after correctly figuring aloud, "three sixes are eighteen; three fours are twelve," he appears to lose the mental image of the eight for which he substitutes a one.

There is no word deafness, and he understands all that is said to him. In general he complies exactly with any oral instructions, but has difficulty when told to place the thumb of the right hand in contact with the third finger of the right hand. Later he executes several similar tests with the thumbs and fingers of the two hands, but seems to experience a little difficulty in thinking out the instructions—he then executes them promptly.

Written instructions (to place the right hand on the left ear, pinching the chin with the left hand, etc.) are promptly executed. He read the headlines from newspapers correctly, but in a broken disconnected manner.

There is difficulty in spelling words, especially the longer ones. "European" is spelled "Euroupean" and he does not recognize the error. He also fails to recognize the error when the word is written out for him. He correctly spells such names as Holmes, Haines, Haynes, which are given him as visualizing tests. He promptly gives rhyming words (pay, sleigh, ray, etc). His manner is at times a little bizarre and extravagant.

August 8, 1919: The patient is more fluent than when seen two days ago. He engages in more protracted conversation, though with frequent delays, during which he searches for missing words, also with frequent mispronunciation. In his paraphasia he usually pronounces the initial syllable or consonant correctly. In endeavoring to say that he is having less difficulty with speech he finds himself checked at the word "difficulty;" he says, "di-dis-difficulty."

There is no evidence of dysarthria, and when he fails to pronounce a word he looks perplexed as though he had difficulty in recalling it. He says, "I can't think of what I like to think of. Then I supply. See?" When told to think of the Lord's Prayer to himself and to indicate when finished, he indicates its completion in twenty-eight seconds; thus finishing almost simultaneously with the examiner.

He cannot recall the lines taught him. "A little learning is a dangerous thing; drink deep, or taste not the Pierian Spring." He says he cannot recall the third word. The following words are written out for him and he is told that one of the words supplies the omission: "Beer, tiredness, knowledge, warfare, love." He selects the word "knowledge," but says this is not correct and brings a book in which the couplet is printed. He then demonstrated that the word is "learning." When asked to write the couplet, he does so with little hesitancy, seeming only to be puzzled about the words "thing" and "spring." He then studies the words carefully before adding the final "g".

Asked to read from the paper, he does so a little slowly and cautiously. He pronounces the word "game" as "dame" and then corrects himself. At the word "threatened" he says, "the" then reads the word correctly. The word "statements" he reads with marked pause between the syllables. "Congress" he pronounces as "college." "Protesting" he gives as "potesting," and he does not correct this error. He states that reading is difficult because of blurring of the eyes; that there is more marked blurring on the left side. Here he says "left" and confirms his meaning with a gesture of the left hand. He says it is very "tite-tiresome" for him to "go through it" (to go on reading).

When asked to recite the couplet mentioned above he begins, "A little something is a dangerous thing." "It is the third rule, road, word, that tea-teases me." Later he says, "bring deep" for "drink deep." In a subsequent conversation he says "cattle" for "capital" (capital letter); then corrects himself. In speaking of his aphasia, he asks, "Don't you think it will wear out? I really nee- it, need it, in my procession-profession."

He whistles the tunes of "Old Kentucky Home," "A Perfect Day," and "America" correctly.

The following surnames are pronounced for him and he spells them orally thus: Fitz (giving Fitts as an alternative spelling), Phelps, Cochran, Sorrenson (also Sorrensen), Spaulding, Spencer (also Spenser), Sprague, Pratt, Prescott, Profitt. Asked to give the capitals in the following names, he spells them as follows: McCormack, MacFarland, McPherson, McCumber, McPhee and McGinnity. The name Cornelius, he spells as "Corneilus" and then adds, "I think." During the testing with these names he is given the surname "Primeaux" and told that the name is French. He spells it "Primo"; then adds, "Unless you want to take it the easy way." When asked what the easy way would be, he again spells it "Primo" (apparently there is momentary confusion).

August 11, 1919: The patient shows further improvement in speech, and at times uses rather long sentences, or several consecutive, short sentences, without error. It is noted that he flounders less with difficult words and seldom makes more than one mispronunciation of a difficult word before he correctly articulates it. He frequently pauses for an instant before a word (suggesting a temporary inability to recall it) and then pronounces it correctly. Thus he says that he is "taking it—easy," and that he is a "little bit—better."

Quite frequently he makes several false starts at a sentence, changing and rechanging the construction, in order to introduce the words that he is able to command and apparently to avoid words that are lost to him by amnesia.

Circumlocutions are quite evident in his conversation. When apparently endeavoring to express the idea that he is talking to give me an opportunity for observation, he says, "I was trying to talk about the thing to give you the range; I was trying to catch the play." When apparently endeavoring to say, "Let me see," he says, "Well, I may see."

During the conversation the following words are recorded as giving the patient difficulty. They are pronounced correctly at the second attempt; the initial attempt is given in parentheses: ride (road); county (ca—); fingers (figures); years (yeal); efforts (eck); states (stages); practically (practi—); syllable (syllas).

For circuit he says "circitit." He says "these two thumbs" for "these two fingers," then corrects himself. He says "consolidization" for "consolidation." When the word "consolidization" is pronounced for him five minutes later and he is asked to point out the error he promptly says that it should be "consolidation." He still pauses at the word "drops" and it seems probable that certain definite words cause him uniform difficulty. He states that he can speak no better when alone than when talking to other people.

He still complains of dizziness and blurring of the eyes (especially in the left visual field). There is still numbness of the index and middle fingers of the right hand, but this tends to be limited now to the tips of the fingers. No headache is admitted.

April 11, 1920: About the middle of February, 1920, he began to complain of severe neuralgic pains in the back of the neck. He is said to have been able to talk well and to go about without evidencing impairment in voluntary movements. Because of the pain he was put to bed and perspiration induced by a hot pack. The pain suddenly lessened, but on the following morning, on attempting to speak, he was able only to mumble inarticulately. Complaint was made that vision was weak and misty, while mentally he wandered in a delirious manner; there were transitory and fragmentary delusions, such as the belief that his father had been married while absent on a visit to the city. Marked restlessness with rolling from side to side supervened. On attempting to walk he was unable to maintain himself, staggered and required to be held in order to avoid falling.

At this time he was lying in bed indicating a recognition of those who came into view. He replied responsively but articulation was so markedly impaired as to be scarcely intelligible. There was reason to believe that he understood all that was said to him, as indicated by his compliance with requests involving responses of both psychic and motor form. The right hand failed in associated movements, and there was definite dysmetria in either arm. On standing there was much incoordination and some propulsive effort, requiring support to avoid falling. Vertigo was not admitted; there was no headache or vomiting, and he complained of nothing. Sphincteric control was maintained. The brow was evenly wrinkled; the face drew more to the left side on emotion, volitional attempts being about equal. The tongue was protruded in the middle line without tremor or atrophy. Other voluntary movements evidenced no alteration in power but much lowering in coordination and especially in movements requiring precision. Direct percussion of the skull over the frontal areas revealed a distinct difference of the two sides in that a high-pitched note was constant on the left, while a dull, "cracked-pot" note, somewhat fluid in character, obtained on the right side.

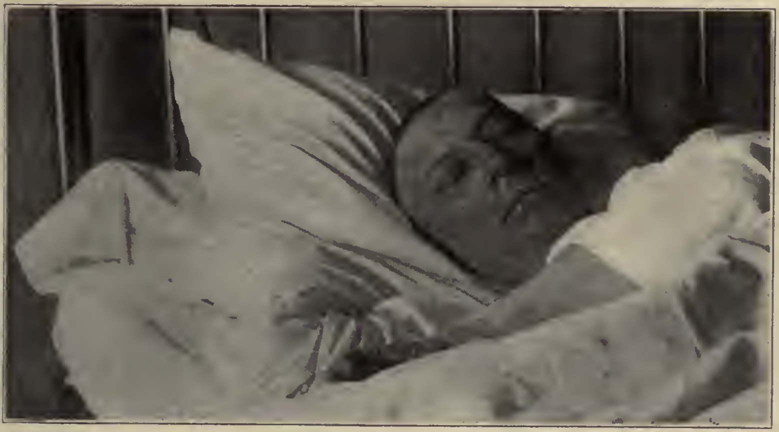

Fig. 3.—Hernia of the frontal lobe following the primary decompression.

Roentgenographic examination showed a distinct shadow in the left frontal area, extending backward to the central fissure. Roentgenograms of the teeth indicated considerable pyorrhea and peridental abscess formation.

Reflexes: All of the deep reflexes were present and about normal in degree and equal on the two sides. The superficial reflexes showed no deviation from the normal, both plantar reflexes being flexor in type and the abdominal reflexes being prompt and equal.

Eyes: All external ocular motion was prompt; convergence was well accomplished, and no nystagmus was evident. The pupils were equal and promptly responsive to light; the right was slightly irregular in contour. The optic fundi were apparently edematous, with very moderate swelling—somewhat less than on previous observation—and no distention of the capillaries.

Hearing: This sense was not impaired for the speaking voice.

April 17, 1920: A decompression operation was done by elevating an osteoplastic flap over the left frontal area; considerable pressure with distention. Insertion of a probe was followed by collapse and drainage of clear fluid. The wound was closed without drainage.

May 12, 1920: A secondary operation was performed under ether anesthesia, and the previous wound was reopened; there was considerable hernia of the brain, especially in the anterior portion of the skull opening. Resistance at a depth of one and three-fourths inches, which later proved to be the wall of a cyst, was encountered. A wick type of drain was placed and carried outward external to the pericranium, the dura was closed about the drain and the scalp brought together in the usual manner. The time required for the operation was one hour; the general condition of the patient was good. The hernia at the site of the operation recurred with considerable distention, which could now be relieved by lumbar drainage.

There followed a gradual decline, more pronounced weakness of the right side of the face, with increasing unconsciousness and death Aug. 2, 1920. There were no convulsions at any time.

Necropsy.—Three hours after death necropsy examination was performed by Dr. Ward Burdick. Permission to examine the brain only was obtained. On removing the calvarium firm adhesions were found under the site of the operation. The convolutions showed no abnormality other than slight injection of the capillaries. The meninges were clear. The right ventricle was somewhat dilated. A cystic cavity was found underlying the second and third frontal convolutions of the left side at about the junction of the middle and posterior third, measuring 4 cm. anterioposteriorly and 2 cm. at its smallest diameter. The cavity was filled with broken-down pultaceous matter and communicated with the lateral ventricle, which was somewhat distended with fluid. Small sections of what appeared to be a fairly defined limiting zone or membrane were retained for examination. Sections of the brain failed to reveal abnormality elsewhere. In this cavity communicating with the ventricle was found a silk ligature drain which was placed at the time of operation.

Pathologic Report.—(By Dr. Harry Gauss). Two pieces of tissue, about 1 cm. each, were received for examination. These consisted chiefly of gray, with some white, substance. The white substance terminated in a grayish-brown membrane-like band of about the thickness of a hair. The tissues had been hardened in liquor formaldehydi for about a week. Preparation for examination included the usual washing, dehydrating, imbedding in paraffin and staining with hematoxylin and eosin. The essential pathologic alterations consisted of miliary punctate hemorrhages, edema, round cell infiltration and neurologic proliferation, These are best described in detail under the several headings:

Hemorrhages: Small miliary punctate hemorrhages were seen in the pyramidal layers of the gray substance. These were round and sharply limited; they measured from 100 to 300 microns and were located chiefly about small blood vessels.

Edema: Throughout the cyst wall, also in that portion of the white substance lying between the wall and the gray substance, as well as in the lamina multiformis of the latter, there were numerous clear distended lymphoid spaces which were extracellular and extravascular. These were numerous and varied from 5 to 30 microns in size.

Round Cell Infiltration and Neuroglia Proliferation: The cyst wall was composed principally of infiltrating small round cells and neuroglial tissue which appeared to have undergone moderate proliferation. The infiltrating cells were almost entirely of the small lymphoid type, although a few polymorphonuclear cells were present. The zone of white cells appeared to form a definite band along the white substance and, generally, did not invade the gray substance, except in a few small areas in which it extended into the lamina multiformis.

These inflammatory reactions indicate a chronic process of benign type. Pathologic diagnosis: A serous cyst of the frontal lobe of the brain.

CONCLUSIONS

It seems obvious from the evidence furnished by the study of this and other frontal lesions that subcortical lesions of the left frontal lobe, and especially the second and third convolution, are likely to be associated with disturbance in movement as proceeding from a preconceived purpose (apraxia), when occurring in a right-handed person. It would appear that progression posteriorly might give rise to disturbance in speech or writing as purposed (apractic aphasia or agraphia) and finally dysarthria. Localized spasm followed by paresis would suggest involvement of the central convolution, which may become generalized, especially if the callosum is included. Incoordination and ataxia may be associated and attributed to mesial extension into the elements of origin of the frontocerebellar tract. (It will be observed that no spatial disorientation was found in the case presented at any time prior to supervention of coma; the knowledge of the patient's surroundings in the hospital and at home was clearly evident at all times.)

Vomiting, headache and optic neuritis may be absent in subcortical lesions of the frontal lobe, especially in the absence of encroachment on the membranes. Unilateral optic atrophy with papilledema of the contralateral nerve is to be interpreted as proceeding from the inferior surface, although bilateral papilledema may occur as a result of rapidly increasing intracranial tension induced by frontal growths as well as by those occurring elsewhere.

325 Mack Building.

A CONTRIBUTION TO THE HISTOPATHOLOGY OF PORENCEPHALUS *

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Porencephalus is always of interest, partly because of the striking disproportion between the great loss of brain substance and the mild degree of functional disturbance. Unfortunately, however, almost all studies of such brain lesions have been limited to gross anatomic observations. It is hoped that this study adds a few facts to our knowledge of the histopathology of porencephalus. Incidentally it emphasizes the close relationship of such defects to the inflammatory encephalitides.

The term porencephalus was first adopted by Heschl,[1] in 1859, to designate such defects in the brain substance as are characterized by cavity formation in the cerebral hemispheres. Such cavities, to be within the limits of his definition, must communicate either with the subarachnoid or ventricular space. Moreover, the defect might be so pronounced as to lead to intercommunication between the subarachnoid space and the ventricular cavity.

Heschl assumed that this loss of brain substance was the result of faulty development. Some injury had occurred during the intra-uterine life to the anlage of certain parts of the brain cortex, resulting in failure to develop, leading also to malformation of remote and dependent fiber tracts. He drew his conclusions from gross anatomic studies of a fairly large number of porencephalic defects.

Later [2] he admitted that porencephalus might be due to a regressive, destructive process occasioned by occlusion of cerebral vessels. He found histologic evidence of degenerative changes, which he interpreted as being vascular in origin and perhaps syphilitic in character.

Kundrat,[3] in 1882, made a further study of porencephalic brains. He reexamined all of Heschl's specimens and added about twelve of his own. His observations, limited mainly to gross anatomic studies, can be summarized as follows:

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* From the Department of Pathology, Mount Sinai Hospital, New York.

* Work carried out during the tenure of a Fellowship in neuropathology.

* Presented before the New York Neurological Society, April 5, 1921.

[1] Heschl: Gehirndefect und Hydrocephalus, Prag. Vrtljschr f. pract. Heilk. **61**:1859.

[2] Heschl: Neue Fälle von Porencephalie, Jubelaeums band der Vrtljhrschr. **100**:40, 1868.

[3] Kundrat: Die Porencephalie, Eine anatomische Studie (monograph).

1. Most of the porencephalic defects are congenital, although infrequently they may be acquired later.

2. Hemorrhage, thrombosis, embolism, violent uterine contractions, hydrocephalus, anemia and psychic disturbances occurring to the mother during the period of pregnancy, are etiologic factors.

3. The porencephalic loss may be so pronounced that the life of the individual is early terminated, or the destructive process, extensive as it may be, not involving any vital centers, permits the individual to mature, although physical and not infrequently mental deficiencies may be striking.

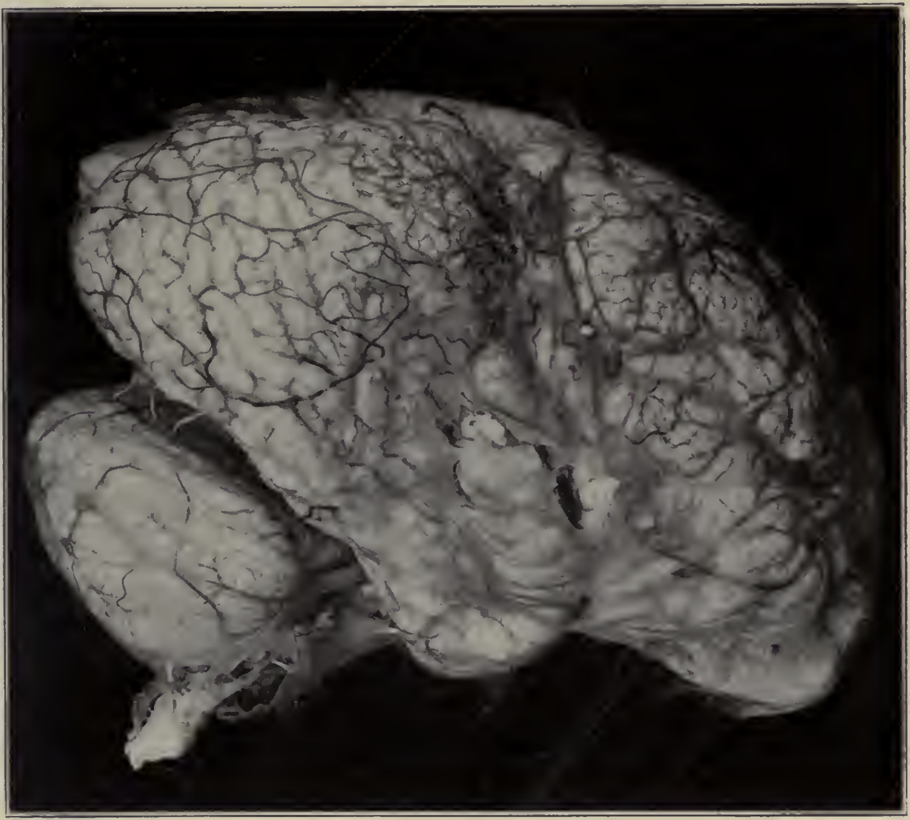

Fig. 1.—Right surface of the brain, showing thickening of the leptomeninges, atrophy of the parieto-occipital lobes, and loss of normal cerebral topography. The irregularly stippled and corrugated surface is particularly noticeable in the region of the parieto-occipital lobes.

4. Destruction or atrophy of part of a hemisphere may be compensated for by relative hypertrophy of the opposite hemisphere.

The main contribution of Kundrat was extension of the term so as to admit other cerebral malformations, which, while not presenting typical cavity formation, are nevertheless characterized by extensive loss of brain substance and by other pathologic features similar to those recognized in the typical form of Heschl. Another suggestion of doubtful value is that "anemic encaphalitis" is responsible for the defect in instances showing no evidence of trauma or changes in the blood vessels. He finds an analogy in the ischemic softening of senile persons without demonstrable changes in the cerebral vessels.

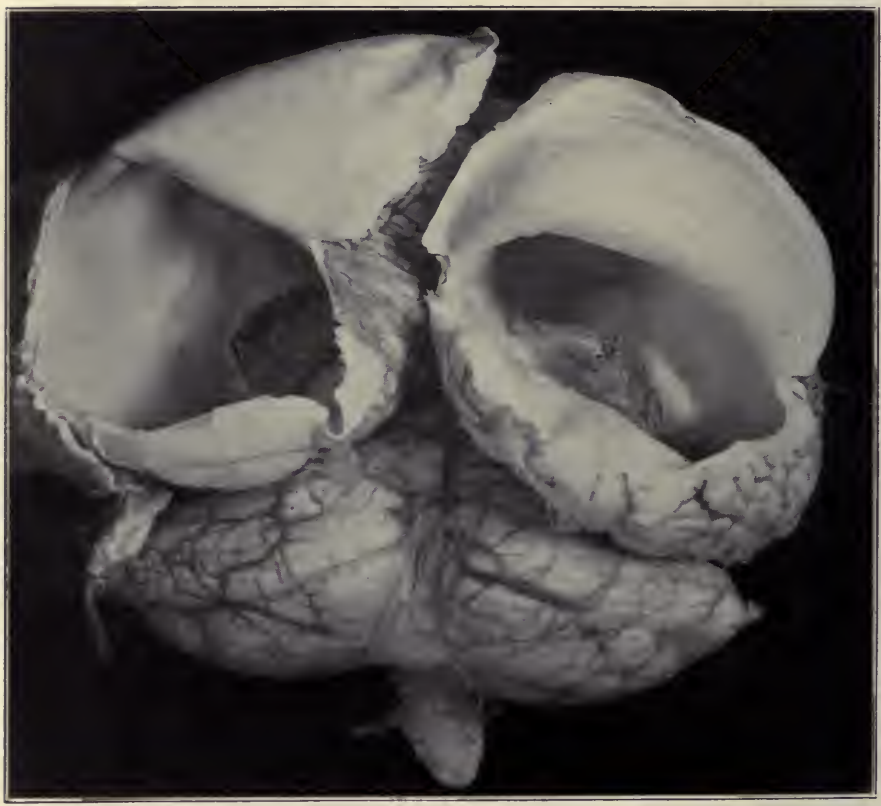

Fig. 2.—Extreme distention of the lateral ventricles and marked thinning of the cerebral hemispheres.

Soon after Kundrat, Schultze[4] suggested that porencephalus may be due to infectious encephalitis. He criticizes Kundrat for failure to study his material microscopically and denies the possibility of anemic encephalitis. He found microscopic evidence of inflammatory changes, probably of infectious origin. He further concluded that the destructive changes began during intra-uterine life.

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[4] Schultze, F.: Beitrag zur Lehre von den Angebornen Hirndefecten (Porencephalie), 58 Versammlung Deutscher Naturforscher und Aerzte in Strassburg, 1885.

That prenatal encephalitis is possible is shown by the observations of Virchow,[5] who established the existence of congenital encephalitis as the result of an infection in the mother. He says: "It is conceivable that such encephalic and myelitic affections are not always fatal and that instances occur where healing takes place, although this fact is yet to be established by further research. . . . It is most probable

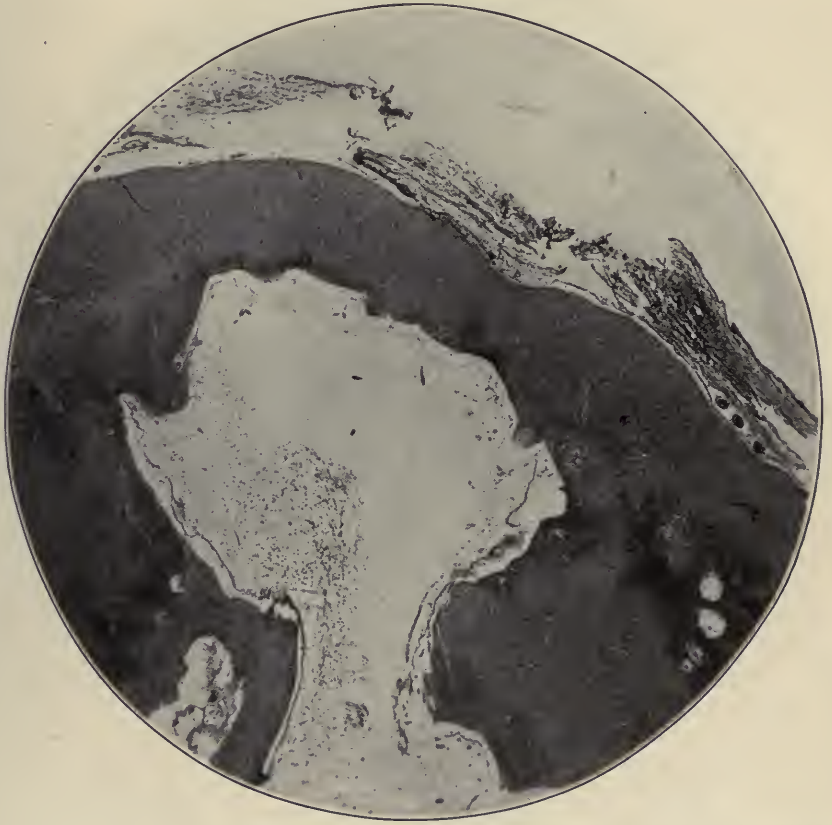

Fig. 3.—A porencephalic defect opening into the lateral ventricle. Smaller defects are seen in the vicitnity of the porencephalic cavity. The pia-arachnoid is thickened and fibrous in structure. Photomicrograph; × 17.

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[5] Virchow, R: Congenitale Encephalitis und Myelitis, Virchows Arch. f. path. Anat. **38**:129, 1867.

that many cases of idiopathic and deuteropathic paralyses of the infant and many instances of idiocy can be traced back to such encephalitic changes."

Strümpell[6] studied acute encephalitis in the infant, clinically and anatomically, and concluded that many porenecphalic defects are due to encephalitis.

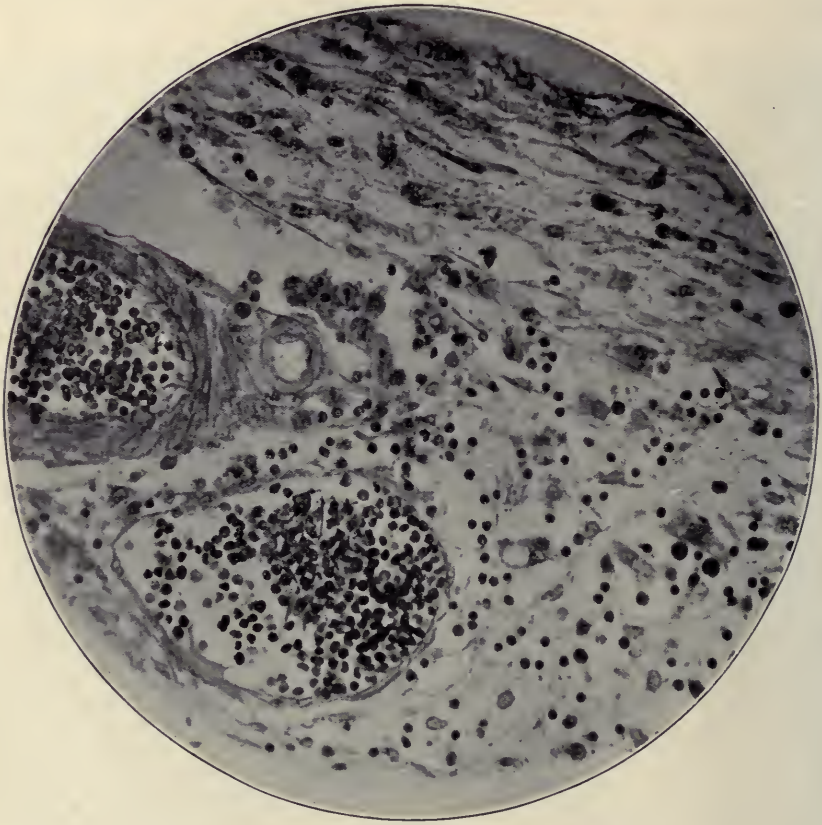

Fig. 4.—Section from the leptomeninges of the central gyrus of the frontal lobe, showing distention and infiltration of the pia-arachnoid space; vacuolated macrophages, small lymphocytes, large mononucleated leukocytes and fibroblasts. The vessels are distended and engorged with blood. The adventitia is thickened and infiltrated. Photomicrograph; × 300.

More recently Limbeck,[7] who studied four cases of porencephalus, has supported the views of Strümpell.

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[6] Strümpell: Ueber die Acute Encephalitis der Kinder, Jahrb. f. Kinderh., 1884, vol. 22.

[7] Limbeck: Zur Kentness der Encephalitis congenita in ihrer Beziehrng zur Porencephalie, Ztschr f. Heilk. **7**:1886.

Sachs and Peterson[8] say: "Porencephalus is a secondary condition and although much has been written on the subject, we know little of its origin. We are willing to concede that some of the many cases of atrophy or sclerosis may have been due to polioencephalitis, but it

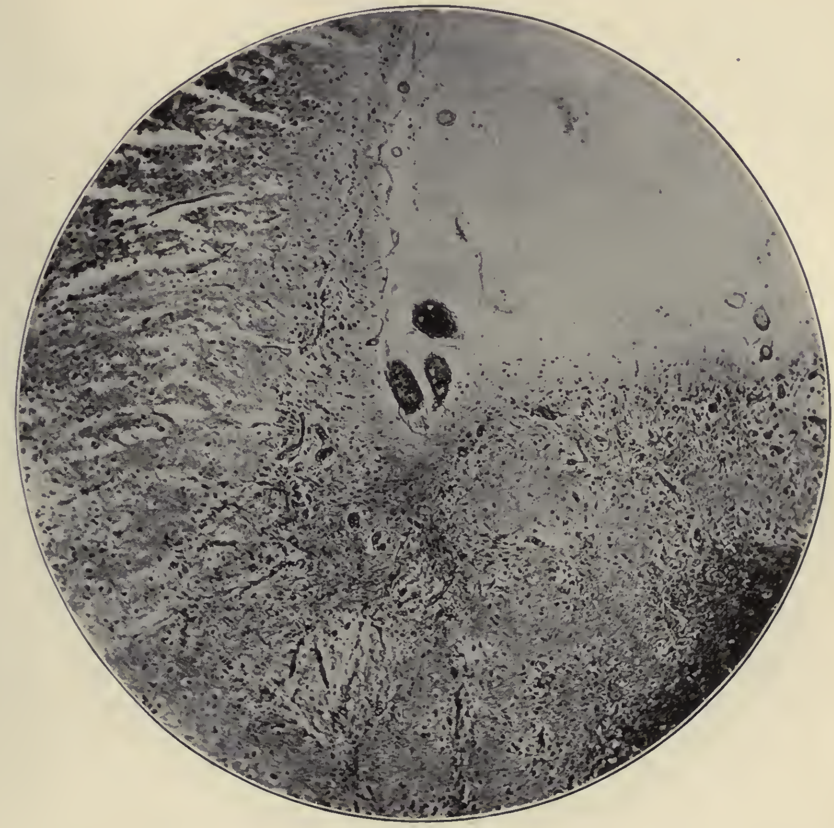

Fig. 5.—Note the marked thickening and fibrous character of the pia-arachnoid covering the parietal lobe. The underlying cerebral cortex is replaced by sclerotic patches. While the vessels on the surface are distended and engorged with blood, the vessels in the cortex are obliterated and represented by fibrous bands. Photomicrograph; × 70; Bielschowsky-Alzheimer-Mann stain.

is unfortunate for Strümpell's theory that all of the necropsies after death have shown other conditions but not encephalitis."

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[8] Sachs and Peterson: A Study of Cerebral Palsies of Early Life based upon an Analysis of One Hundred and Forty Cases, J. Ment. & Nerv. Dis. **17**:319, 1890.

CASE REPORT

History.—G. N., 11 months of age, the first child, was born after a full term pregnancy and normal delivery. There was no evidence of syphilis in the parents, who gave a negative blood Wassermann reaction. At birth the patient was well formed, and he developed normally until the age of 4 months when it was noted that he did not progress mentally and acted as though he were blind and deaf. Aside from a small head, the child showed no physical maldevelopment.

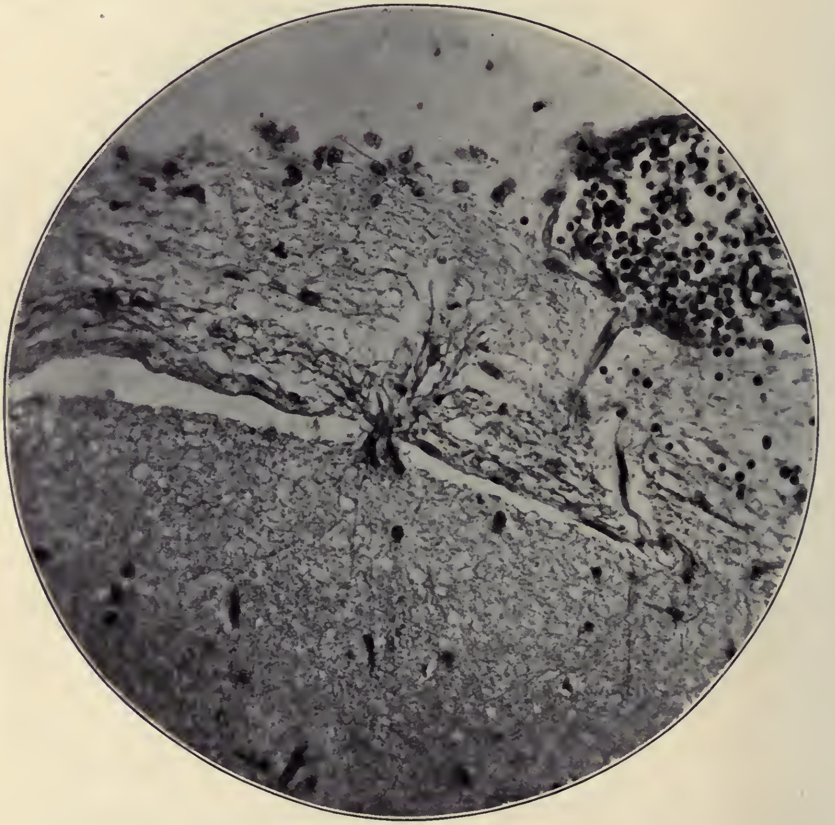

Fig. 6.—Glial cells are seen sending out their processes into the pia-arcahnoid, bridging over the sub-arachnoid space. Photomicrograph; × 300.

Examination.—On admission to Mount Sinai Hospital the following findings were recorded:

Child well nourished and well developed. Head small; fontanelles closed. Pupils regular, react to light and accommodation no nystagnus. Upper extremities spastic and in flexion. Lower extremities spastic; all deep reflexes exaggerated. Temperature irregular ranging from 99.5 to 102 F.

Treatment and Course.—The diagnosis was congenital microcephalus, and bilateral subtemporal decompression was advised, which was performed four days later. After operation cerebrospinal fluid escaped from the wound, but the patient's condition was unchanged. A high variable temperature continued. On the eighth day the child had several attacks of generalized convulsions, grew rapidly worse and died the same day.

Necropsy.—Heart, spleen, liver, and suprarenal glands showed no gross or microscopic lesions. The lungs, with the exception of some emphysema, were normal. The kidneys showed a mild degree of cloudy swelling.

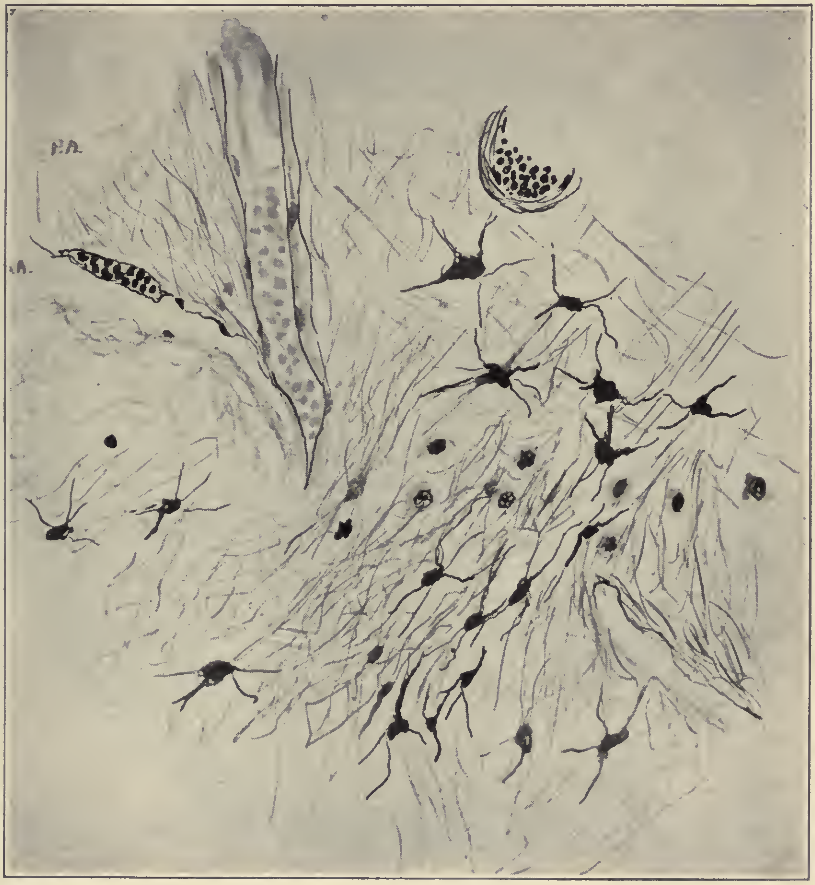

Fig. 7.—Glia cells are seen in large numbers streaming into the pia-arachnoid alongside vessels that are still patent in their meningeal course, but hardly penetrating the cerebral cortex. Camera lucida drawing (Zeiss ocular 6 × objective 3).

The brain was rather small and apparently somewhat collapsed due to postoperative escape of cerebrospinal fluid. The meninges over the dorsolateral surfaces of the cerebral hemispheres were thickened in some areas and firmly adherent to the underlying cortex; in other areas they were greatly distended by fluid. The meninges at the base were normal.

On palpation the hemispheres fluctuated due to marked increase of cerebrospinal fluid in the ventricles, and particularly because of thinness of the walls. Right Hemisphere: The sylvian fissure was well developed at the base but rather wide and shallow in its posterior part. It lodged the middle cerebral artery which ran its normal course over the frontal lobe and for some extent over the parietal. The frontal and parietal opercula were poorly developed, exposing the island of Reil, while the temporal operculum was normal. The convolutions of the frontal lobe could be made out although the fissures were shallow and the gyri extremely flat. The precentral gyrus was fairly well defined from the postcentral area by a deep central sulcus. The gyri in front of the precentral sulcus were indistinct because of meningeal thickening, adhesions and atrophy of the gyri. The postcentral gyrus was poorly defined and the parieto-occipital lobes were markedly atrophied. Here the normal markings of the cortex were entirely lost. Instead there was a stippled, corrugated, granulated hob-nail surface. The vessels were freely movable

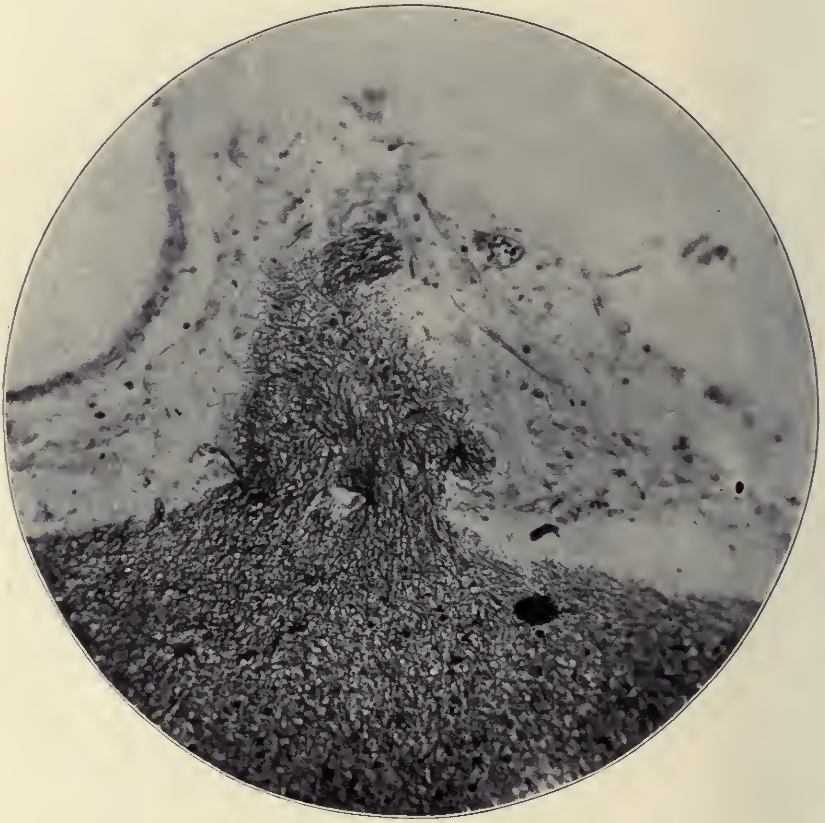

Fig. 8.—Section from the pons. Here a more massive migration of glial elements leads to protrusion of neuroglia into the pia-arachnoid. Photomicrograph; × 180.

over the surface (Fig. 1). On incision the cortex of the occipito-frontal lobe was found to be only about 2 to 3 mm. thick. At the occipital pole it was reduced to parchment-like thinness (Figs. 2 and 3).

Left Hemisphere: This was similar to the right. The sylvian fissure was well defined but shallow, exposing a poorly developed island of Reil. The middle cerebral artery was in the sylvian fissure and appeared to be patent throughout. There was no first or second frontal convolution but the third was present. The portion corresponding to the first two presented a granular, stippled surface covered in places by thickened meninges. The first temporal gyrus was well developed; the second and third were absent, the latter being replaced by the granular cortical structure. The gyri on the base of the temporal lobe were absent, but the hippocampal gyrus was normal. The parieto-occipital lobes were similar to those on the opposite side. The orbital surface of the frontal lobe was normal.

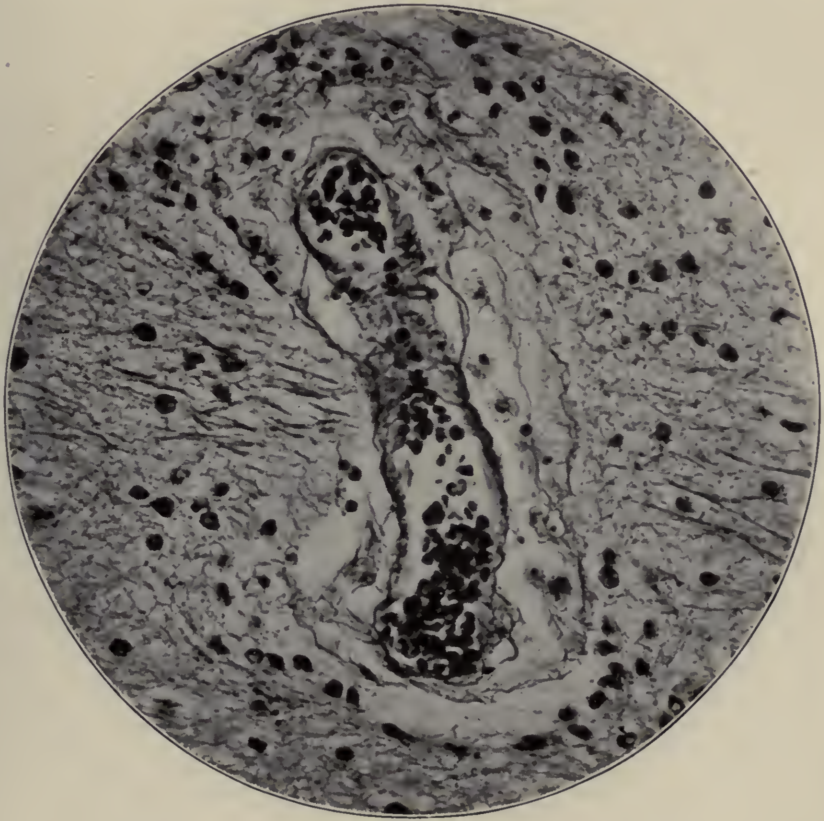

Fig. 9.—Section from the precentral convolution of frontal lobe showing marked distention of the adventitial space of the blood vessel. The granular (Gitter) cells found in the adventitial space point to a still active destructive process. Photomicrograph; × 400.

The cranial nerves were normal.

The ventricles were markedly distended in all their divisions and with the exception of the frontal lobes left nothing but a shell of the cerebral hemisphere. The basal ganglions were well developed; the corpus callosum was present; the septum pellucidum was thin, stretched and fenestrated. The mesencephalon, pons, medulla and cerebellum appeared to be normal. The hypophysis was small, the sella shallow; the blood sinuses and middle ear were normal.

Microscopic Findings.—The thickening of membranes over the dorso-lateral surface varied. Thus, over the frontal lobes where the leptomeninges were edematous, the subarachnoid space was distended and infiltrated with elements characteristic of a subacute inflammatory process. The changes in the pia-arachnoid, pointing to a more or less recent inflammatory process, were characterized by macrophages with or without enclosures and with or without vacuolated cytoplasm. Endothelial cells grouped in fairly large numbers about blood vessels constituted an important feature in the cellular infiltration. They were recognized by their large vesicular nuclei, poorly staining chromatin material and abundance of cytoplasm. Small lymphocytes were also seen

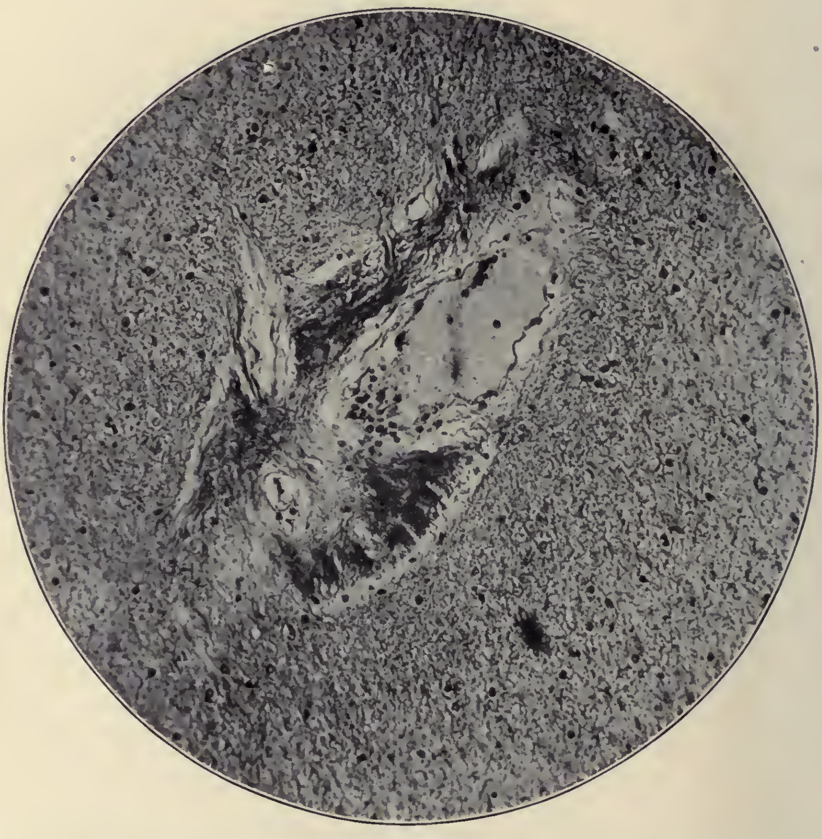

Fig. 10.—Vessel surrounded by a zone of dense gliosis. The adventitial wall of the vessel is infiltrated by small lymphocytes. Photomicrograph; × 200.

in fairly large numbers. Fibroblasts giving off delicate processes formed in places a delicate network. Plasma cells with eccentric nuclei, deep staining chromatin and faintly staining cytoplasm were rare. The blood vessels were much dilated and congested and showed thickening of the adventitia (Fig. 4).

A striking change in the leptomeninges was seen in the wide parieto-occipital area. The transition from the subacute inflammatory process in the frontal meninges to the chronic fibrous thickening over the parieto-occipital area was abrupt. About 5 mm. posterior to the central fissure the leptomeninges were represented by a thick fibrous structure poor in cellular elements in which it was impossible to distinguish the pia from the arachnoid (Fig. 5). In some places extremely short pial processes could be traced into the cortex where blood vessels penetrated, but the majority of cortical vessels were apparently cut off by the meningeal cicatrization; others were strangulated by glial changes at a point just below their entrance into the cortex and in their deeper course were often obliterated and converted into a narrow band of connective tissue.

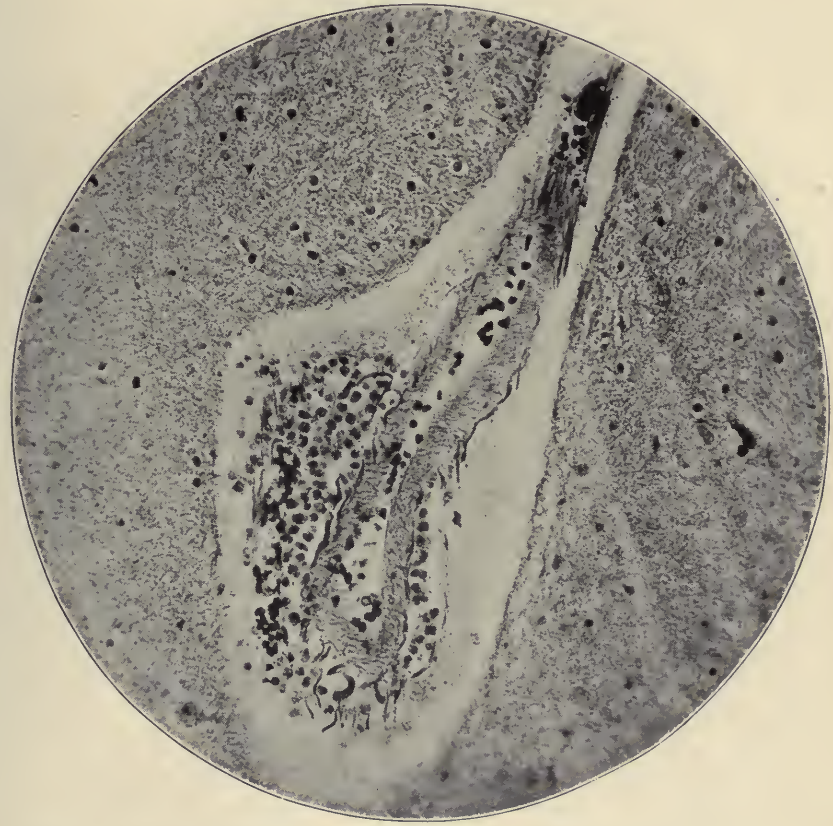

Fig. 11.—Section from the midbrain at the level of the inferior quadrigeminate bodies showing extensive adventitial infiltration of the vessels with mononuclear elements. Photomicrograph; × 240.

Strangulation and obliteration of blood vessels led to sclerotic changes in the cortical and subcortical substance. These zones of sclerosis corresponded to the areas supplied by obliterated vessels (Fig. 5).

There was a transition between these two types of meningeal alteration over the temporal lobe, particularly over the third temporal convolution. There was increase in fibrous elements with moderate mononuclear cell infiltration. Of interest was the fusion of the cortex with the pia-arachnoid alongside closed or partially closed vessels brought about by a most unusual migration of glial elements into the pia-arachnoid. In this way the boundary between the mesodermal pia-arachnoid and ectodermal glia-supporting tissue was effaced. Thus individual glial cells could be seen migrating into the pia-arachnoid, their processes extending over the subarachnoid space while the cell bodies were still in the peripheral zone of the cortex. Also larger groups of glial cells were noted streaming into the pia-arachnoid membrane, alongside a partially obliterated blood cell (Figs. 6 and 7).

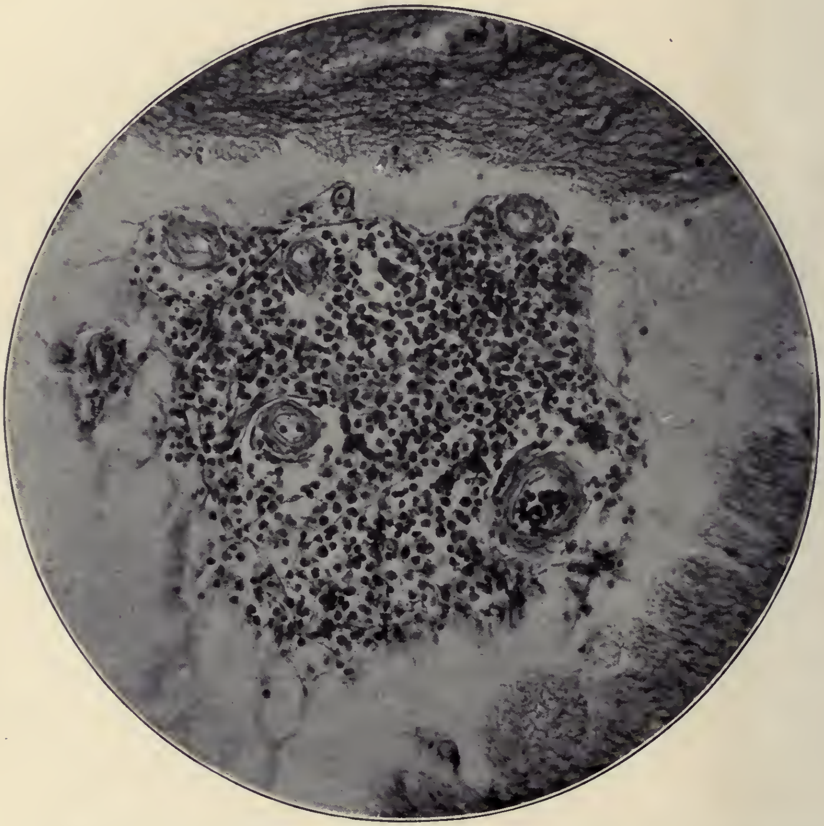

Fig. 12.—Section from the pons, showing a group of small vessels enveloped by a large mass of small lymphocytes. The adventitial walls of the vessels appear to be thickened. Photomicrograph; × 240.

This reaction of glial cells to anemic conditions resulting from obliteration of blood vessels is thus clearly demonstrated and is in full accord with the frequently expressed opinion that glial elements usually react to circulatory disturbances in the central nervous system by proliferation.

This feature is most pronounced where the inflammatory process is most recent, as in the pons. There massive migration of glial elements gives rise to herniation of neuroglia into the pia-arachnoid (Fig. 8). Such a process was thought and is still considered by many writers to be impossible. But Saxer,[9] in studying syringomyelia, called attention to glial infiltration of the pia. He even described the transformation of glial cells, on reaching the pia-arachnoid, into macrophages and other phagocytic cells. Also Strauss[10] has demonstrated involvement of the pia by an infiltrative subependymal glioma.

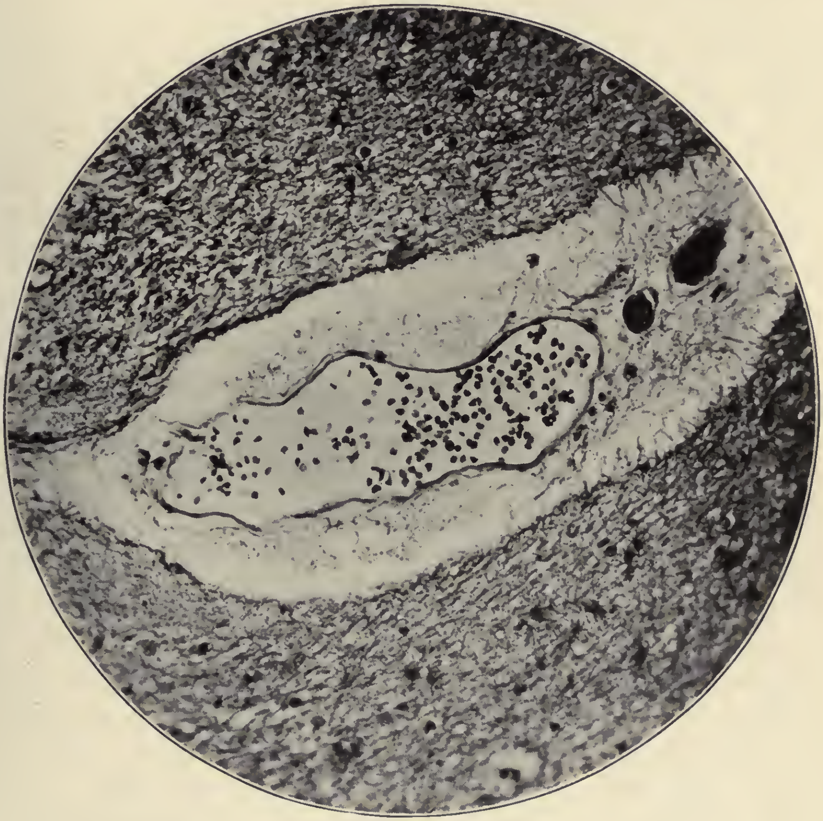

Fig. 13.—Pons; vessels with distended perivascular space filled with serous exudate and a few lymphocytes. Photomicrograph; × 240.

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[9] Saxer: Anatomische Beitrage zur Kentness der sogenannten Syringomyelie, Ziegler's Beitr. z. pathologischen Anatomie, **20**:332.

[10] Strauss, I.: A Glioma Infiltrating the Pial Membrane, New York M. J. 1908, p. 1079.

Apparently the inflammatory process was primary where it now presents the more chronic features. Next, the inflammatory process involved the temporal lobe and finally the frontal lobes.

Cortex: The lesions of the cerebral cortex corresponded to the chronicity and character of the disease in the meninges. In the parieto-occipital lobes, where the meninges were fibrous, the cortex and subcortical substance were only a thin layer of scar tissue. Only here and there one could see a poorly preserved ganglion cell (Fig. 5).

The cortex of the frontal lobe presented an entirely different picture. Here many ganglionic elements were preserved, but the large motor cells were few in number and badly damaged. Important was the evidence of recent degenerative and inflammatory change. Large collections of fat globules crowding the adventitial space pointed to an active destructive process in the brain substance. Vessels with perivascular small round cell infiltration and with gitter cells crowding the adventitial space were further evidence of progressing inflammation (Fig. 9). Of particular interest were the vessels surrounded by zones of marked gliosis (Fig. 10) as well as wedge shaped zones of sclerosis resulting from the strangulation of the blood vessels by the cicatrizing pia-arachnoid.

In the mesencephalon at the level of the trochlear nerve was evidence of recent trouble—vessels with well defined adventitial infiltration. The cells found in the adventitial space were mostly small mononuclears, with a few plasma and endothelial cells (Fig. 11). In the pons was an area in which a group of small, probably newly formed blood vessels was enveloped by relatively large collections of mononuclear cells (Fig. 12); also vessels with serous exudate into the distended adventitial spaces (Fig. 13) and a limited number of perivascular hemorrhages were found.

Spinal Cord: Though it appeared that the ventral gray columns on one side were poorer in ganglion cells than on the other, it was difficult to establish a primary inflammatory process in this area.

COMMENT

The observations recorded in the foregoing present abundant evidence of an encephalitic process early in the life of the infant. The extensive destruction of the cerebral cortex and eleven months of the post-natal life not marred by any illness lead to the conclusion that the destructive process, resulting in the porencephalic defect, must have begun in the latter part of the intra-uterine life period and remained active and slowly progressive after birth.

That the physical development of the child was practically uninfluenced by the enormous loss of brain structure is most striking. This is best explained by the lack of early disease in the basal ganglions, midbrain, pons and medulla.

The opinion that the process was inflammatory and not traumatic or developmental is well borne out by the microscopic findings. Chronic and subacute lesions were found in the leptomeninges and underlying hemispheres, subacute and more recent lesions in the midbrain, pons and medulla. Wherever the meningeal lesions were of the chronic fibrous type, complete strangulation of blood vessels resulted in diffuse and pronounced atrophy of the brain. The parenchyma was replaced by wide sclerotic zones, with fibrous bands.

In the temporal region, the pia-arachnoid was in process of fibrinous organization, shown by the presence of many fibroblasts and numerous mononuclear elements. Findings here indicate that wherever circulatory disturbances occur in the brain tissue, the destruction of brain parenchyma is accompanied by proliferation of glial elements. The thickening and cicatrization of the pia-arachnoid lead to strangulation of the blood vessels, producing ischemic zones of softening and sclerosis in which proliferation of glial elements and migration along closed blood vessels in the direction of the pia-arachnoid membrane are readily seen. This unusual process is of interest because it may help to a better understanding of changes that occur in multiple sclerosis, gliosis and scar formation in the central nervous system.

The histologic findings in the more recent lesions, especially in the region of the frontal lobe (mononuclear leukocytes, fibroblasts, plasma cells and macrophages) speak for a still active pathologic process. So probably the disease, started in the parieto-occipital region, spread to the temporal lobe, thence to the frontal region and finally to the mesencephalon, pons and medulla.

Repeated attempts to find the causative agent of the encephalitic process failed. Nevertheless the changes in the brain were clearly inflammatory, placing the case in the Strümpell group, so aptly named by Sachs—polio-encephalitis corticalis cerebri.

CONCLUSIONS

1. This case, although characterized by marked atrophy and diffuse sclerosis, can be considered one of porencephalus.

2. The atrophy and porencephalic defect were due to an inflammatory process, beginning as a meningo encephalitis in the parieto-occipital lobes.

3. The meningitic process led to strangulation of the blood vessels, producing anemia, degeneration of the parenchyma, proliferation of glial elements and finally to scar formation in the cortex.

4. Glia cells proliferate whenever blood vessels are occluded and migrate along the closed vessels toward the seat of active inflammatory process.

5. The migration of glial elements into the pia-arachnoid is well established by the findings recorded and supported by the observations of Saxer and Strauss.

6. The inflammatory process, primary in the meninges of the parieto-occipital lobes, slowly progressed to the temporal, then frontal lobes and finally to the midbrain and hind brain.

7. The strangulation of blood vessels causing sclerotic changes in this case is an important finding in relation to the pathology of disseminated sclerosis.

ARTIFICIAL PNEUMORACHIS IN THE TREATMENT OF ACUTE INFECTIONS OF THE MENINGES

A PRELIMINARY REPORT*

EDWARD AFFLECK SHARP, M.D.
BUFFALO, N. Y.

Injection of air or oxygen into the cerebral ventricles and subarachnoid space has become a recognized and valuable procedure in neurologic and surgical diagnosis.

As a therapeutic measure, injection of oxygen or air into the subarachnoid space by means of lumbar puncture, producing an artificial pneumorachis, has been found of considerable value in the treatment of some of the acute infections of the meninges.

During a small epidemic of meningococcus meningitis in 1916-1917 a few cases were found in which the purulent spinal fluid was obtained with difficulty owing to adhesions or blocking of the vertebral canal. Injections of half strength normal saline solution diluted the fluid and broke up adhesions, permitting a free flow of the purulent fluid. In some of these cases oxygen was injected with the result that an additional quantity of purulent fluid could be evacuated before injecting the antimeningococcus serum. In other cases the air or oxygen was used alone with the same favorable results in evacuating the fluid.

The technic employed is the ordinary procedure of lumbar puncture with the patient lying on the side. After removal of as much fluid as will flow through the needle, the oxygen is injected by means of a 20 c.c. Luer syringe. The syringe is filled by allowing the pressure from the oxygen tank to force back the plunger of the syringe. Sterile gauze is interposed between the gas cylinder and the syringe during the filling process. The oxygen is injected slowly, using from 5 to 10 c.c. as measured on the syringe. Removal of the syringe allows the gas and fluid to escape in a frothy mixture. Injection of the gas is repeated and the head and shoulders slightly elevated to allow the gas to reach the cerebral ventricles. This results in additional fluid escaping when the syringe is removed. In the meningococcus cases the serum is injected after this flushing-out process.

In these cases the oxygen probably acts only as a mechanical agent in displacing the fluid and opening up secluded pockets of adhesions.

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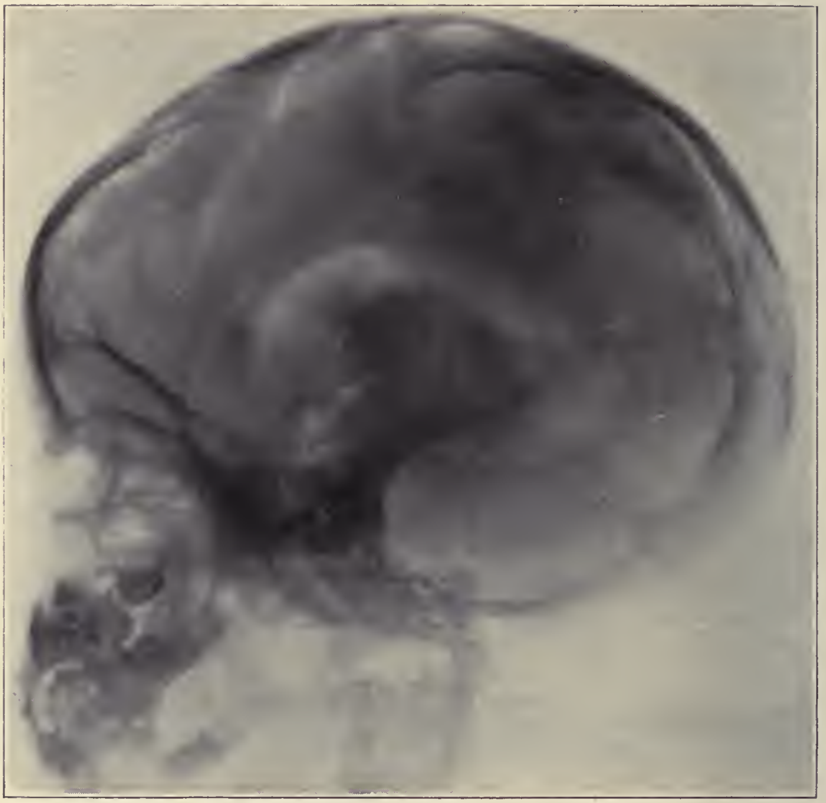
* Read at the Forty-Seventh Annual Meeting of the American Neurological Association, Atlantic City, N. J., June, 1921.

That the oxygen actually reaches the ventricles is shown by the increased amount of fluid forced out, the changed percussion note over the lateral ventricles, and the visible proof seen in the illustration in which the cerebral ventricular space is shown filled with oxygen which was injected by lumbar puncture.

The illustration shows that the gas has spread over the cerebral cortex in the subarachnoid space and fills the cisterna magna. Radiograms are thus of service in showing the patency of the foramina of communication between the ventricles and the subarachnoid space and in localizing pockets of purulent fluid walled off by adhesions.



Ventriculogram showing lateral, third and fourth ventricles filled with gas. Oxygen was injected by lumbar puncture.

THERAPEUTIC EFFECT OF OXYGEN AND OTHER GASES

The therapeutic effect of oxygen or other gases in contact with the meninges or ventricular ependyma is difficult to estimate. The fact that different gases are absorbed at varying intervals of time suggests that some therapeutic value might be placed in the use of them. No harm has resulted in repeated injections of oxygen in the meningococcus cases. The absorption is fairly rapid as fresh amounts can be injected daily at the times of giving the serum. The patients are usually too ill to move to the roentgen-ray room for pictures at regular intervals.

In tuberculous meningitis repeated injections of oxygen has appeared to produce favorable results in a few cases. Whether it was the contact of the oxygen with the meninges or the repeated lumbar puncture with forced drainage that gave favorable results in three cases is impossible to determine. In three of twelve cases of tuberculous meningitis a complete recovery resulted, and the patients are well after an interval of a year or more. While the actual diagnosis of tuberculous meningitis may be questioned in these three cases, owing to absence of tubercle bacilli in the spinal fluid, the cases were in all other respects clinically tuberculous meningitis. The spinal fluid in each case showed a high cell count of lymphocytes, clear fluid and escaping with considerable pressure, a well-formed fibrin pellicle on standing; increase in globulin and albumin and a negative Wassermann reaction. One of the children had been under treatment for several years with Pott's disease; another had scrofulous glands in the neck which had been operated on; while in the third case the mother had a tuberculous history, although the child showed no other clinical signs of infection, aside from the meningitis.

None of the patients showing tubercle bacilli in the spinal fluid have recovered although in most of them the oxygen injections produced temporary improvement and even return of consciousness for a few hours.

In lethargic encephalitis less value should be placed on any form of intraspinal treatment owing to the deep-seated location of the lesions in most cases. One case, however, of the epileptic-maniacal type in which the lesion was probably a meningo-encephalitis responded to the lumbar puncture withdrawal of the spinal fluid and oxygen injection. In one other case of the lethargic type recovery from the deep lethargy followed promptly the injection of 15 c.c. of oxygen.

The average amount of oxygen injected has been from 10 to 15 c. c. The amount actually left in the spinal canal is difficult to estimate as a considerable portion escapes with the frothy fluid. In the case shown in the ventriculogram, the amount retained in the spinal canal and ventricles was over 20 c.c.

No ill effects or alarming symptoms have resulted from the oxygen injections. When the larger amounts of gas are retained in the spinal canal it is better to avoid too sudden elevation of the head and filling the cerebral subarachnoid and ventricular cavities too suddenly. A slight dyspnea, due to bulbar irritation, has occurred when the filling has been too rapid, but the symptoms have not been alarming.

During the past five years a total of sixty-four cases have been treated by the intraspinal injection of air or oxygen, as shown in the table.

DISEASES IN WHICH INTRASPINAL INJECTION OF AIR OR OXYGEN HAS BEEN USED DURING PAST FIVE YEARS

Disease	Recovered	Died	Total
Meningococcus meningitis	23	7	30
Pneumococcus meningitis	0	5	5
Streptococcus meningitis	0	4	4
Typhoid meningitis	0	1	1
Tuberculous meningitis	3	9	12
Lethargic encephalitis	8	2	10
Poliomyelitis (acute)	2	0	2
Total	36	28	64

Of the total of sixty-four patients treated by artificial pneumorachis twenty-eight have died. At first glance this does not appear to differ materially from the usual statistics in such cases. In the meningococcus meningitis cases only the severe and apparently unfavorable cases were treated by this method. The mortality of 23 per cent. compares very favorably with the statistics of all cases of meningococcus infection in which the antimeningococcus serum is used alone.

The mechanical effect of the gas in opening secluded pockets of infection has undoubtedly prevented relapses as none of the patients who have recovered have had a recurrence such as occasionally occurs in other cases.

The experience in the few cases enumerated shows that oxygen may safely be injected in the spinal canal and that it acts as a mechanical and possibly as a therapeutic agent in the acute meningitides.

481 Franklin Street.

DISCUSSION

Dr. M. Allen Starr of New York asked whether in the literature or in Dr. Sharp's experience there were any fatal cases and what the proportion of deaths was.

Dr. Ernest Sachs of St. Louis asked Dr. Sharp whether in these cases of epidemic meningitis the patient had first had the Flexner serum and whether at the time when the oxygen was injected the organisms had disappeared from the fluid, because sometimes even when it is difficult to get some of the fluid out, the organisms have disappeared, and it raised the question as to whether the oxygen itself offered any therapeutic help. It seemed to him that the type of case in which oxygen might help would be with an anaerobic organism, as Dr. Ayer had said; using oxygen with an aerobic organism, which the meningococcus is, would hardly seem to inhibit its growth and act therapeutically; it may act mechanically.

Dr. Colin K. Russel of Montreal asked whether the two patients with poliomyelitis quoted by Dr. Sharp died or recovered with the usual paralysis.

Dr. James B. Ayer of Boston thought the subject of pneumorachicentesis was of much interest. The procedure had of course been carried out by Dandy, and in Boston Dr. Cruthers had safely used it at the Children's Hospital for diagnosis. Concerning its use as a therapeutic procedure, he did not feel that it had been demonstrated that air or oxygen is of specific benefit in meningitis, and it should be shown that it is the air, and not simply irrigation, that is of value. If the three cases of meningitis mentioned were actually tuberculous, which unfortunately was not proved, then the success of this method must be admitted, in that such types almost always are fatal. It appeared that in the hands of Dr. Sharp the method had proved safe, and should therefore be further employed, although with great care and with due attention to pressure changes during the procedure.

Dr. Edward A. Sharp of Buffalo, in closing, stated that in some of the cases pneumoracentesis had been performed merely as a last resort. In 1916, two patients died, but they were practically moribund at the time. The injection of oxygen had no effect in hastening the end; in fact, in none of the cases had the oxygen injections been responsible for a fatal outcome. The value of the oxygen is probably mechanical in its effect of removing more fluid.

Since hearing Dr. Ayer's paper last year the idea of using the cisterna magna puncture to inject the oxygen in those cases in which adhesions prevent the fluid reaching the lower spinal dural sac seemed practicable.

Dr. Sharp said that oxygen probably had very little, if any, effect in destroying the germs, the effect being largely mechanical. The contact of the oxygen with the inflammatory area might have a formidable therapeutic effect by increasing the vascular supply.

Opening the abdomen in cases of tuberculous peritonitis had occasionally been followed by long remissions in the symptoms, and the favorable results had been attributed to the contact of the peritoneum with the air or oxygen. It was possible that a similar theory might be applied to the meninges.

Replying to Dr. Sachs' question, Dr. Sharp said the Flexner serum was used in all the meningococcus cases. In the severe cases the irrigation was employed at the first injection. When the fluid was thick the irrigation was done with saline solution and then followed by the oxygen. At the hospital patients had done better when the subarachnoid space had been fairly free from pus before the injection of Flexner's serum. The saline irrigation and the oxygen had apparently had no effect in lessening the activity of the serum. In all cases the serum was used early. As stated by Dr. Sachs, when the fluid became thick and difficult to get out the organisms were not found.

Previous to using the oxygen method at the Ernest Wende Hospital, a number of patients had relapses after apparently clearing up, due to pockets of pus which were not reached by the serum. With the oxygen method, breaking up adhesions, the relapses have not occurred.

Dr. Sharp's experience in the poliomyelitis cases had been the same as that mentioned by Dr. Russel. The two patients mentioned were in the hospital while the work was in the experimental stage, and the injections were performed as a routine procedure. The effect of removing the larger amounts of fluid by displacement with oxygen was apparently favorable.

A SYNDROME OF THE VISUOPSYCHIC CORTICAL AREA—BASED ON STABILE HALLUCINATIONS AND DEFECTIVE VISUAL ASSOCIATION IN A SANE PERSON *

H. H. HOPPE, A.M., M.D.
CINCINNATI

The discussion of this subject presupposes that in normal persons the stimuli perceived by the visuosensory center are transmitted to the visuopsychic areas, are there compared with previous visual pictures and are recognized as similar or dissimilar. There they are stored, but the manner in which they are stored is unknown.

Memory pictures stored in the visuopsychic center can be recalled to the field of consciousness either by the will or by stimuli coming from other cortical areas through the association tracts. Stimuli coming from without through the visual organs can also recall these memory pictures, and the flow of these stimuli is always from the visuosensory to the visuopsychic area. Never under normal conditions can the process be reversed. Stimuli from the visuopsychic or other cortical areas cannot stimulate the visuosensory areas. If this were not true, we could never be certain of the objective reality of our visual impressions.

Under abnormal conditions this flow of stimuli is reversed—memory pictures stored in the visuopsychic areas, stimulate the visuosensory area and are then projected into space. They are then known as hallucinations.

In considering syndromes of the visual area of the brain, we must distinguish between lesions of the visuosensory area and lesions of the remainder of the occipital cortex, namely, the visuopsychic area. The classical sign of the former is simple hemianopsia unassociated with other signs and of the latter, mind-blindness.

We must also distinguish between lesions which are cortical and those which are subcortical, between irritative lesions and destructive. Lesions, however, which we are called on to localize are not always strictly localized to either the sensory or the psychic areas, and the same lesion may be irritative first and destructive later on. Thus, irritative lesions of the calcerine area may cause simple or elementary hallucinations, such as flashes of light or color, which precede the development of the hemianopsia. If the lesion is more widespread we may have more complex hallucinations projected into the failing field

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* Read at the Forty-Seventh Annual Meeting of the American Neurological Association, Atlantic City, N. J., June, 1921.

of vision, but with cortical destruction of the visuosensory area, the only sign is blindness. The projection of visual hallucinations presupposes an anatomically normal visuosensory area.

Hemianopsia, alexia and agraphia are not, strictly speaking, an occipital lobe syndrome, but are caused by a subcortical destruction of the association tracts beneath the gyrus angularis.

An irritative lesion of the visuopsychic area may be one-sided and may show itself in the appositive visual field as one-sided hallucinations. This may occur occasionally with intact vision, as in Peterson's well-known case. Cases of hallucination in hemianopsia, in which the visions are projected into the blind field, are proof of the foregoing assertion.

Mind-blindness, which is the typical sign of a destructive lesion, requires a destruction of both visuopsychic areas or their association tracts from the visuosensory area. I wish to call your attention now to another syndrome of the visuopsychic area, which is probably caused by an irritative lesion. This syndrome presents these three factors: (1) persistent stabile visual hallucinations with intellect, speech and general memory intact, (2) normal visual perception, and (3) disturbed visual association. The occurrence of stabile visual hallucinations in persons who are mentally normal is not frequent. Instances which come under our observation are considered sufficiently rare to excite comment, and we are apt to remember them as cases of unusual interest. In these persons single objects are usually seen and then only on a single occasion. I do not refer now to epileptics with visual aura or to cases of hemicrania.

A woman physician, a patient of mine, with a tendency to hysterics would have attacks of great fear produced by the vision of a white cat walking across her consultation room. Nearly everyone knows of some person or has heard of some one who has had a vision.

Guerney[1] asked 27,737 persons whether they had ever had an hallucination and obtained affirmative answers from 12 per cent. of those questioned. The impression, however, is made that these persons had experienced the illusion or hallucination only once.

Stearns,[1] in his study of 500 cases of mental disease, observed in the Boston Psychopathic Hospital, says that hallucinations are rarely observed in sane persons even though they are psychopaths. In his group of 500 cases, twenty-four were demented and had arteriosclerosis; only three had hallucinations, and two of these were insane.

Hallucinations occurred least frequently in the group of those not insane, as only 6 per cent., four out of sixty-five, were affected. Two were hysteric patients who had had one hallucination each, and the other two were children with pavor nocturnus.

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1. Stearns: J. Nerv. & Ment. Dis., 1915.

I do not hesitate to place a rather unusual clinical case on record because: 1. A person may have visual hallucinations of distinct groups in the form of definite pictures extending over a long period of time and still be of normal mind. 2. The case offers a definite visual memory field syndrome which may have a definite localizing value.

There are comparatively few observations on record of persistent visual hallucinations in persons not insane. Most of these occur in cases of hemianopsia with hallucinations in the blind field, or in cases in which fleeting hallucinations preceded the occurrence of blindness in destructive lesions.

AUTHOR'S CASE

History.—E. H. F., a widower, aged 91, at the age of 61 had a right-sided hemiplegia with aphasia. He recovered completely in three months. About one year ago he had a stone removed from the bladder by the suprapubic route. He lost his fortune at the age of 75, but made another. Within the last six years the patient has put into operation a new large enterprise, which is successful. He shows good business judgment and is daily engaged in directing large enterprises.

Present Illness.—About four weeks ago the patient began to have "visions." These occurred when he was wide awake, either when sitting in his library or when resting in his bedroom. These "visions" occurred daily and annoyed the patient. They produced no emotional reaction, and they were recognized by the patient as not being real.

These visual hallucinations can be divided into four groups:

1. The patient on looking out of his window at a neighboring lawn always sees a large red brick house. As soon as he fixes his attention on the house, it begins to move toward him, comes up to the window and then disappears.

2. On looking out of the window the patient sees a lake with ships on it. One of these ships begins to sail toward the window and when it comes abreast of the window it disappears. Both of the hallucinations can be shut out by closing the blinds.

3. Groups of men, white and black, swarm into the library from the hall door—at times the figures are whole, at others, there are only heads. Again the heads and faces appear distorted—the noses are at times missing, heads seem to be moving about, severed from their trunks. These figures are always in motion.

4. Well dressed women walk into his bedroom. They pay no attention to him, walk past the foot of his bed to a cheval mirror, arrange hat and veil, adjust their clothing and then walk out of the room. The apparitions are always silent—they never speak and never pay any attention to the patient.

These visual hallucinations never vary from these groups. They are not present when the patient is motoring or when he goes about his warehouses, offices or stock farm. They never produce any emotional reaction except annoyance and the fear that he might be considered insane. He knows that they are not real. They occur only when he is wide awake—never in the half conscious state preceding or following sleep. The patient does not dream.

Physical Examination.—The patient was physically well. The arteries at the wrist were palpable, but soft. The blood pressure was: diastolic 95, systolic 140. The aortic valve sound was only slightly accentuated. Pupillary reaction was normal. Ophthalmoscopic examination showed that the disk, retina and vision were normal. There was no defect in the function of any of the cranial nerves. The upper extremities were normal for sensation, muscular power and reflexes. The lower extremities were normal, there was no change in the reflexes, muscular power was normal. There was no trace of the hemiplegic attack of thirty years ago. Urinalysis was negative. His intellect was normal. He was a keen business man and personally attended to large affairs. He showed a normal interest in local, national and international affairs. He was oriented for time and place, was very cleanly in his person and careful of his dress and appearance. His memory for events both recent and past was good. There was no speech defect. The patient could read and understand what he read. There was no amnesic or optic aphasia.

Visual Tests.—There was no loss of vision, the patient could see and recognize any object which was shown him, knew the use of it, and could describe it. If shown pictures of single objects he recognized them and named the colors correctly. He was shown pictures, for instance, of men and fish and made no mistake as to the nature and color of the object.

Association Visual Memory Test.—The patient was shown many pictures and was not able to interpret the meaning of them. When asked to describe the picture in words he would read the advertisement or explanation printed below, he could not grasp the story told by the picture. In one picture, which was an advertisement of the Cragmoor Sanitarium, he pointed out the buildings but could not recognize the mountain range, finally saying that he saw another building where the mountain range was portrayed. When shown another picture which was used as an advertisement for a breakfast food, he could not interpret the story conveyed by the picture. When shown groups of men in other pictures he was not able to tell what scene the picture was intended to portray. Men, fish or other printed objects, if single, were recognized. In other words, visual memory association was lost. Analytic and synthetic comprehension of pictures was impossible. Stereoscopic vision was normal. The patient was also oriented as to space, recognized his surroundings, was able to go about the house, go out to and enter his automobile, and walk about his extensive farm without any difficulty.

He had no difficulty in placing or recognizing the proper position of objects in space. Spatial orientation of an object seen showed that there was no faulty projection. He was accurate in judging distances of objects in space. Perception of movement and light perception were normal. He was not color blind. Psychic blindness in the ordinary sense was not present, for the patient both named and recognized the nature and use of objects.

CAUSES OF HALLUCINATIONS

This case is reported in order to discuss whether we may not have here a new syndrome of an irritative lesion of the visuopsychic area, composed of: (1) persistent stabile visual hallucinations; (2) normal visual perception for simple objects; (3) disturbed visual association, and (4) normal mentality.

This patient had no lesion of the visuosensory area, nor were the association tract or tracts between the visuosensory area and the visuopsychic area broken down, for there was no mind-blindness, no loss of orientation in space and he had no trouble in judging the distance of objects from the body. His color perception and color sense were good.

There was no optic aphasia, no alexia and no hemianopsia which would indicate that the subcortical tracts coming to the occipital lobe from the region of the gyrus angularis were intact.

The patient did not recognize his hallucinations as real, they did not affect his judgment and emotions, which would indicate that his remaining cortex and their association tracts were intact and normal in function.

His inability to explain the meaning of pictures showed a lack of normal visual memory for the purpose of association of previous memory pictures. Visual memory, synthetic and analytic, was defective. This is a function of the visuopsychic area.

Hence, reasoning by exclusion, the clinical picture is produced by an irritative and perhaps partly destructive lesion of the visuopsychic area caused by senile arteriosclerotic changes. I believe, therefore, that this case proves that we can have a visual syndrome characterized by persistent stabile hallucinations, with normal vision perception, without mind-blindness but with defective visual analysis and association, in a person who is not insane.

Another interesting feature which this clinical case brings out clearly is that a person may have persistent stabile visual hallucinations which may persist over a period of months and not be insane. I believe that the proof of this assertion is made clear by a careful reading of the case.

Most of our knowledge of the genesis of visual hallucinations in sane persons has been obtained from the study of hallucinations in hemianopsia. It is a prerequisite for the occurrence of hallucinations in cases of hemianopsia that the visuosensory area should be normal. The conduction apparatus for vision and the subcortical tracts need not be normal, for visual hallucinations may occur in the blind, where blindness is due to diseases of the conduction tracts. A lesion, therefore, which destroys the conduction tract on one side and at the same time exerts an irritative influence on the visuopsychic area of the occipital cortex can prodce visual hallucinations in the blind field. As there are not many organic lesions which can simultaneously destroy and irritate, cases of this kind are relatively infrequent. Henschen was the first to hold that these cases of hemianoptic hallucinations were not due to lesions of the visuosensory area but to irritative lesions of the visuopsychic area and that they, therefore, have a certain localizing value.

It has long been known that visual hallucination can be caused by destructive lesions of the occipital lobe, but in these cases the visual hallucinations always precede the occurence of half-sided blindness.

Thus Climenko[2] reports a tumor of the occipital lobe with progressive hemianopsia. When the headache was very severe the patient saw many colored animals and objects pass before his eyes. "He had hallucinations of myriads of rapidly moving objects and various colors."

Hallucinations occurring in the hemianoptic field either preceding or following the loss of vision prove that hallucinations can arise from mechanical pressure or from irritative lesions of the visuopsychic area.

Hallucinations always point to an abnormal condition of the central nervous system. This, however, need not necessarily mean a destructive lesion; and while hallucinations usually occur in the insane, their presence is not pathognomonic of insanity even though they persist for weeks and months. The crucial point of difference is what becomes of the hallucinations—Does the consciousness of the individual recognize the hallucination as such or does it accept it as real?

The result of the hallucination depends on the sum total of consciousness and the integrity of the other sensori-psychic areas. If the latter are normal, the visual error or hallucination is corrected. Consciousness, attention and correct functioning of the other senses enable people to bring proof that the hallucination is not an objective sensory impression.

Fleschig[3] explains this by saying that if only one sensoripsychic center is involved the individual remains sane, but if various sensory centers together with the posterior association areas are involved the patient becomes confused and is the victim of an hallucinatory psychosis.

DISCUSSION

Dr. Hugh T. Patrick of Chicago asked Dr. Hoppe whether the visions always approached from the same side. He also asked whether the patient was right-handed and in which field the visions occurred.

Dr. M. Allen Starr of New York thought that this was a very interesting case on account of its duration.

He called attention to the fact that hallucinations of shorter duration might occur under the influence of poisons. He had a patient brought to him, a very intelligent Protestant woman, who had awakened one evening with very bright hallucinations of the Virgin Mary with the infant in her arms, appearing at the foot of her bed. She was extremely alarmed and was very much perturbed at this appearance which persisted constantly for four days, giving rise to the suspicion or fear on the part of the family that she was going crazy.

Dr. Starr could, however, find no symptom of mental aberration, no loss of memory, no delusional state, merely the persistence of this marked hallucination of vision. It was not attended by hallucinations of hearing and was not accompanied by any other symptoms, with the exception of a great sense of fear and alarm. He noticed that there was a great dilatation of the pupils, dryness of the throat and of the skin, and on inquiry found that her family physician had prescribed for her an ordinary purgative or laxative pill. He had written the prescription, which was shown to him en masse, putting in 3 grains of belladonna, to be divided into twelve pills, and the probability was that the druggist in putting it up, managed to get all 3 grains of belladonna into one pill, and that the hallucination was due to poisoning by belladonna. The event proved to be so, because as the days went on, the dilatation of the pupils subsided, the dryness of the mouth and skin subsided, and she gradually lost this particular hallucination and was well. She has had no recurrence for six or seven years. Therefore, a hallucination may last a considerable time—in this particular case four days continuously—simply as the result of accidental poisoning.

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[2] Climenko: J. Nerv. & Ment. Dis., 1915, p. 828.

[3] Fleschig: Gehirn und Leele.

Dr. H. H. Hoppe of Cincinnati, in closing said that he had found, in looking through the literature on the subject, that none of the patients was subjected to visual association tests; mention was made that the individuals had visual hallucinations only.

Answering Dr. Patrick, he said it was not a case of hemianopic hallucination, as the man's vision was normal. The patient was right-handed.

News and Comment

SECOND ANNUAL MEETING OF THE ASSOCIATION FOR RESEARCH IN NERVOUS AND MENTAL DISEASES

The second annual meeting of the Association for Research in Nervous and Mental Diseases will be held at Hotel Commodore, New York City, Dec. 28 and 29, 1921.

PROGRAM

MULTIPLE SCLEROSIS

Presidential Address....................................Dr. Walter Timme

SYMPTOMATOLOGY

General Symptomatology..................................Dr. Bernard Sachs
Special Symptomatology Study...
.........Drs. J. W. McConnell, Williams B. Cadwalader, George Wilson
Hyperkinetic Motor Phenomena.........................Dr. Frederick Tilney
The Location of Lesions with Respect to Symptoms........Dr. E. W. Taylor
The Spinal Fluid in Multiple Sclerosis..................Dr. James B. Ayer
Aural and Vestibular Manifestations....................Dr. Isidore Friesner
Ocular Manifestations...................................Dr. Ward A. Holden
Psychotic Symptoms.................Dr. Sanger Brown, 2d, Dr. T. K. Davis
Psychologic Factors.................................Dr. Smith Ely Jelliffe
Clinical Endocrine Studies.......Dr. Walter M. Kraus, Dr. Irving H. Pardee

STUDIES IN ETIOLOGY

General Etiology..................................Dr. Lewellys F. Barker
Bacteriologic Studies in the Pathogenesis of Multiple Sclerosis..........
....Dr. Oscar Teague
Pathologic Studies in the Pathogenesis of Multiple Sclerosis...........
....Dr. George B. Hassin
Endocrine Studies, Macroscopic and Microscopic.........................
..........................Dr. Hubert S. Howe, Dr. Myrtelle M. Canavan

STUDIES IN PATHOLOGY

Pathologic Considerations in Multiple Sclerosis.........Dr. William G. Spiller
Axis Cylinder Changes in Multiple Sclerosis................Dr. J. H. Leiner

INCIDENCE AND STATISTICS

Geologic and Ethnologic Features..Dr. C. B. Davenport, Dr. C. B. Esterbrook
Multiple Sclerosis as Revealed by the Draft.............Dr. Pearce Bailey
Statistical Data......................................Dr. I. S. Wechsler
Bibliography..Dr. Frederick Tilney

Abstracts from Current Literature

ACUTE CIRCUMSCRIBED EDEMA AND RELATED CONDITIONS.

H. Quincke, Med. Klin. **17**:675 (June 5) 1921.

In this rather extensive article the author reviews the subject of angioneurotic edema and allied conditions. It is his belief that the disease is not as uncommon as usually supposed, owing to the fact that the usual case is transient, not serious, and rarely requires the services of a physician.

Quincke has observed from sixty to seventy cases. In the last thirty-six cases 50 per cent. occurred in males and 50 per cent. in females. The more complicated and atypical cases appeared in the females. The youngest patient was 13, the oldest 68. The majority of patients had their first attack when between 21 and 30 years of age. The average duration of the disease was five years, the minimum a single attack, the maximum thirty years. The length of the single attack varied from a few hours to six days, but usually lasted a day and a half. The eruption may be so fleeting as to go unobserved.

The length of intervals between attacks varies greatly—from twenty-four hours to years. At times the attacks occur at irregular intervals. They may occur in series with a long period of remission. Two such cases are briefly reported, the first patient having had two attacks in twenty-one years, the second patient two attacks in four years, the last attack consising of a series occurring daily, usually most severe between the hours of 7 and 11 p. m. In another case the interval was twenty-six and eighteen years, respectively.

Usually with increasing age the attacks of edema become lighter and more infrequent, or may stop entirely. Periodicity is common; the attacks occur not uncommonly at certain times of the month, on certain days, on rising in the morning, after the noon-day meal, etc. A close relation to migraine, epilepsy, hydrops genu intermittens, certain colics, types of erythema and neuralgias is noted in the paroxysmal type of the attacks.

The etiology of the disturbance is not yet clear. Menstruation, psychic excitement, fatigue, loss of blood, heavy meat consumption, constipation, articles of diet, such as strawberries, chemicals, such as caffeine, aspirin, etc., are mentioned. One patient gave the cause as sea-bathing, two as warm baths, three as pressure or other mechanical measures; excessive sunshine is also mentioned. Climatic conditions are also referred to, but usually the associated change in the diet plays a more important part. In one of the cases reviewed by the author the cause of the edema was thought to be due to the absorption of toxins as the result of a cholecystitis, the edema disappearing on the removal of the gallbladder.

As a rule, patients having an angioneurotic edema appear healthy. During an attack, except for the local discomfort, little disturbance is noted. In only three cases were any febrile signs noted.

In 39 per cent. of the cases general nervousness was present; 13 per cent. showed a familial tendency to urticaria or acute edema. The majority of patients were anemic and sedentary and reacted well to change in mode of living and exercise. Robust out-of-door persons were, however, also affected.

In Quincke's edema, the swellings vary from 2 to 10 cm. in diameter. The margin is not sharply defined, but blends into the surrounding skin. The color may be unchanged or slightly red, there being no increase in local heat. The edema is firm and does not pit; the patient usually complains of a feeling of fulness, occasionally burning, rarely itching. Besides the skin, the subcutaneous tissue may be involved.

Any grade of hyperemia may be present up to a distinct wheel formation with temperature, itching and pain, but no proportional constitutional symptoms. At times there may be local tenderness before the appearance of the edema. Quincke's edema and urticaria, while usually very distinct conditions, may show any possible admixture. In four cases, urticaria had existed for years prior to the appearance of the circumscribed edema. Urticaria is frequently seen as an associated condition in the familial type of edema. The edema may also be associated with erythema multiforme, erythromelalgia, and Raynaud's disease.

Frequency of involvement of various parts is given: face, 80 per cent.; eyes, 64 per cent.; lips, 53 per cent.; legs, 44 per cent.; hands, 41 per cent.; arms, 25 per cent.; tongue, 22 per cent.; prepuce and scrotum, 20 per cent.; thighs, 19 per cent.; pharynx, 17 per cent.; mouth, 8 per cent.; gastro-intestinal organs, 5 per cent.; esophagus, 3 per cent., and periosteum, 5 per cent.

Occasionally symmetrically situated parts are affected one after the other. There is no rule, however, as any or all of the areas mentioned may be affected in one attack or in succeeding attacks. In no case in this series was the larynx involved; rarely the mouth, pharynx, stomach or intestines are involved, and, if so, only in association with some other region.

Ten cases are cited by the author to illustrate the unusual distribution which the edema may take. Several atypical cases are also reviewed. In one interesting case in this series, the patient, a female, had for months a swelling of the eyelids, first one, then the other, usually occurring in the morning. Finally, under calcium lactate treatment, the condition cleared away. Another case was that of a patient who had swelling of the face for eight months, first diagnosed as erysipelas.

Migraine was found to be frequently associated with Quincke's edema (seven cases). In some cases the headache precipitated the attack, in others no apparent relation was present. In one case of severe migraine which had existed from the age of 10 to 50, there developed at the latter age, with practically every headache, a swelling of the face and lips, or an edema occurred without headache.

Quincke is of the opinion, as is also Bürgi, that migraine is a manifestation of meningeal edema, exactly as angioneurotic edema is a swelling of the skin or mucous membranes. On this basis Quincke believes that one is able to explain the great variety of cases of migraine, and also the numerous cerebral focal signs, as edema of the meninges may occur anywhere as it may on the skin.

In a case of left-sided headache noted by the author, in which the headache lasted for three days, there was an associated twitching of the right side of the face and right arm with weakness, lasting several days, but always passing away. Cases of hemiplegia with migraine have been observed, and Quincke has seen two patients with monoplegias. These cases he believes are due to edema of the pia with pressure or possible associated cortical involvement.

Hydrops genu intermittens, edema of the bursae and tendon sheaths are also discussed by the author. Cerebrospinal fluid exudates are also noted, and the author reports three cases in which, associated with the menstrual period, there occurred a serous meningitis.

Recurrent neuralgias are mentioned as a possible result of edema. One case of recurring sciatica, according to Quincke, was due to localized edema of the lumbar plexus. Recurring myalgias are also due to this cause. In some of the studied cases colloid changes in the muscle were thought to be the cause, but Quincke personally believes that the condition is due to an edema of the intermuscular connective tissue fibers.

Various other types of edema are mentioned by the author under the heading of circumscribed edema. In one case reported by Neuda and Gagstatter, edema of the mucous membrane of the bladder was noted.

Edema due to other causes, such as nephritis, cardiovascular conditions, mechanical reasons, etc., is reviewed by the author. In discussing the physiology of this condition the author reviews several theories based on ion concentration.

The transitory character, the periodicity and the location point to a nervous origin, as do also the usual history of nervousness, familial tendency, psychic emotions and the known similarity to migraine and epilepsy.

Under the heading of treatment the author has little new to offer, diet, hygiene, etc., playing the usual important rôle. Other therapeutic measures are mentioned but none that has been used with uniform success.

Moersch, Rochester, Minn.

THE ANATOMICAL AND CLINICAL SYNDROMES OF THE CORPUS STRIATUM. J. Lhermitte, Neurol. Bull. 3:163 (May) 1921.

The author presents a comprehensive study of this important structure. He insists clearly and logically that there is no evidence to justify the assumption that the corpus striatum is in any sense regressive in structure or function. We have come to our present knowledge of this subject by the anatomicoclinical route; physiologic investigation has been misleading and practically barren of constructive result.

The anatomoclinical syndromes are grouped under three headings: those in which the lesion involves the corpus striatum in its entirety; those which affect the striatum (putamen and caudate nucleus); and those which implicate the pallidum. The pallidum includes not only the two internal segments of the lenticular nucleus, but also, according to Ramsey Hunt, the large-celled neurons scattered over the putamen and caudate nucleus. The pallidal syndrome would appear to be determined not only by destructive or degenerative alterations affecting the globus pallidus, but also by those that reach the pallidal element carried to the interior of the striatum along with the purely striate neurons (striopallidal system). It will be seen that Lhermitte's studies are governed chiefly by histologic considerations.

Under syndromes of the entire corpus striatum the author considers: first, the progressive lenticular degeneration of Kinnier Wilson; second, pseudosclerosis; third, the senile and presenile pseudobulbar syndrome of striate origin which differs from Wilson's disease in the type of destructive process. In the former there is gross vascular alteration; in the latter "even though the loss of nerve and neuroglial elements have a paravascular origin, the lumen of the capillaries, veins and arteries remain quite normal"; fourth, striate syndromes caused by presenile lesions of the striatum and pallidum (C. and O. Vogt); the senile parkinsonian syndrome.

Syndromes of the striatum include: first, the syndrome of Cecile Vogt, with marbled appearance of the striatum; Little's disease, with congenital and regressive spasticity alone; second, dysmyelinization (C. and O. Vogt); general rigidity with progressive terminal athetosis; third, Huntington's chronic progressive chorea, the result of "atrophic cortico-striate degeneration." In this type the author feels that true adiadokokinesis is exhibited, and therefore that this symptom is probably not the exclusive property of cerebellar lesions, but that it should be accorded a place among the striate signs; fourth, the author includes acute lesions of the striatum—Sydenham's chorea. In this and in Group 3 we find diminished muscular tone and the hypertonicity of other striate syndromes is replaced by hypotonicity. Thus a coordinator rôle is assigned to the striate body; fifth, lesions (hemorrhages or softening) in the region of the striatum.

Syndromes of the pallidum bring into the discussion the much mooted question of Parkinson's disease, particularly paralysis agitans according to the views of Ramsey Hunt.

All the diseases of the striate body have certain symptomatic elements in common. They are exclusively motor; there is universal muscular rigidity, clearly distinct from pyramidal spasticity; there is marked diminution of spontaneous voluntary movements. To this are related the disturbances of the normal facial expression and gesturing that accompany language and thought.

The author's conclusions are embodied in his closing sentences. "What appears most clearly when we leave anatomico-clinical syndromes and attempt to picture the functions of the corpus striatum as a whole is that the influence of the purely motor functions of this structure are very different from that of the cortico-bulbo-spinal system (pyramidal system). The latter has to do with the most highly differentiated and delicate movements. They may be considered the most intellectual and consequently the most human. All the more rudimentary functions are carried out by the striate system. They are the older movements of the vertebrate series, and, continuing the metaphor, more animal in character.

"From the physiopathologic point of view, those conditions which bring about more or less complete destruction of the corpus striatum affect functions which are essentially automatic and which are only slightly influenced by the cortex. Walking, the synkinetic movements of defense and of spatial orientation, the mimicry of gesture and facial expression, swallowing and phonation are all functions of an automatic character. It is these automatic functions, long gone from the plane of consciousness, which, as the Vogts pointed out, are affected by disease of the corpus striatum.

"Everything leads us to the general conception that the motor corpus striatum and the sensory optic thalamus correspond to the precentral motor gyrus and the postcentral sensory gyrus which receives conscious sensations. And, further, the corpus striatum and the optic thalamus control the superior automatic motor system, while the cortical centers dominate and control the voluntary motor system."

Strecker, Philadelphia.

THE POST-TRAUMATIC HEADACHE SYNDROME. R. Benon and B. Kerbrat, Schweizer Arch. f. Neurol. u. Psychiat. **8**:184, 1921.

Some time previously Benon called attention to a post-traumatic nervous condition, which he designated by the name traumatic headache or the syndrome of post-traumatic headache. He states that the fundamental and initial symptom of this syndrome is headache, and that the various nervous and psychic manifestations which accompany it are secondary and determined by it. The syndrome may for this reason be separated from post-traumatic neurasthenias and the less severe forms of traumatic dementias. Three case histories are reported:

The first patient was a soldier, 21 years of age, who, in 1914, suffered a cerebral commotion as the result of an explosion of a projectile. He was unconscious for a period of from twenty-four to forty-eight hours. Prolonged treatment at various stations was undertaken with indifferent results. In November, 1916, he was seen by the writers. At this time the fundamental symptom was headache. This was present continuously, but fluctuated in intensity. Violent paroxysms of pain, particularly in the occipital and sincipital regions with radiation toward the temples, occurred. The pain was described by the patient as being like a heavy weight on the head; when the pain became intense it semed as though terrific blows were being struck. During exacerbations, there was vertigo and a profound alteration in character; the patient assumed imperious attitudes, came and went, gesticulated, and was very much agitated, struck at doors, and jostled his comrades. He became fatigued easily, and there was some confusion of ideas and difficulty in remembering. The family history was essentially negative except that his mother was of a nervous temperament. Previously the patient had been rather timid, but alert.

The second patient was a male servant, 16 years of age, who, in 1912, had been kicked in the left frontal region of the head by a horse. At that time a diagnosis of cerebral commotion and fracture of the frontal bone was made. After five days he was able to be up and about. He was examined from time to time until October, 1917. The dominant symptom was headache, which was continuous but fluctuated in intensity. He was exceedingly irritable, struck at objects, spoke in a loud voice, and was rather menacing toward those about him. This agitation would last several hours, at which time he would feel that he would never again be able to live like his comrades. The more he became demoralized, the more violent he would be. With a lessening in intensity of the headache, there was a return of optimism. At times he suffered from nightmares. Although this headache had continued for a period of five years, it had not changed in intensity. The family history was negative except that a sister committed suicide.

The third patient was an infantryman, 31 years of age, who, in September, 1916, suffered a cerebral commotion with multiple contusions from the explosion of a projectile and burial under a mass of débris. Consciousness was lost at once, but returned after an hour. There was extreme ringing in the ears, and great pain in the head and in the chest. His treatment at a number of different hospitals was unsuccessful. The patient was seen by the writers in September, 1917, when he was found suffering from a continuous headache present on awakening and persisting until night. It did not hinder sleep, however. The pain was always great but was subject to violent and intolerable paroxysms. Its location was in the neck, with radiation to the temples. The patient described the sensation as though a machine were in continuous movement in the head. During the paroxysms he was more irritable and at the least contradiction felt pain in the chest and experienced a sense of choking. The family history in this case was negative, except for nervous crises occurring in the mother.

These three observations are typical examples of the post-traumatic headache syndrome. The asthenic manifestations, alterations in character, hypochondriac and melancholic tendencies are all in direct relation to the intensity and violence of the headache. The headache usually has its seat in the occiput and is extremely severe.

Woltman, Rochester, Minn.

SOME PROBLEMS CONFRONTING THE ASSOCIATION. Owen Copp, Am. J. Psychiat. **1**:13 (July) 1921.

This indictment of the present status of mental medicine combined with definite working plans for a bright future is too valuable not to be read by every neurologist and psychiatrist.

The counts in Dr. Copp's indictment are the indifference of the public and physicians to giving mental patients an even chance with sufferers from other forms of illness, the lack of adequate psychiatric instruction in medical schools and the appallingly inadequate facilities for research in mental diseases.

Plans for remedying present conditions are given in considerable detail. They involve prevention through school inspection and the teaching of special groups of children; through the increase in instruction to student physicians. An available agency of prevention as well as treatment is the bringing of patients with mental cases to general hospitals where in the psychopathic wards they can receive emergency care and short and intensive treatment for "mental states not recognized as such by patient or his friends and for other conditions which are curable and not disturbing to the general hospital regime." This is essential in order to get good advice for the mental patient from specialists in all departments of medicine; and it is by the treatment of these specialists that many physical causes can be removed.

There follows an unusual summary of the possibilities of treatment in hospitals. In the first place, there is treatment in the university clinic (capacity 100) or in the endowed private hospital (capacity 100 to 300) of special cases for special purposes and combined with intensive research. In the second place, or as a substitute, classification can be obtained in metropolitan (psychopathic) hospitals (capacity 100 to 200) with rapid turnover of patients and close affiliation with medical schools.

In the third place, there is treatment in the district (state) hospital with a maximum of 2,000 patients, the most important single agency in the treatment of mental disease and so the place at which the "maintenance of the highest standards is imperative." This hospital must as a practical matter not only receive patients directly, study them as do the clinics, but also be equipped to hold patients for indefinite periods and to provide a farm colony. This hospital "will minister to more than 90 per cent. of mental patients throughout the longest illness; will have exclusive opportunity in most cases, so that any deficiency means to the patient proportionate deprivation of the best hope of restoration to health or alleviation of disability. It must strive to meet every requirement of every type of mental hospital because the necessity of every kind of service is sure to arise without possibility of other aid in many cases." This district psychopathic hospital both from the humane and the scientific standpoint must be protected from politics. Its surest safeguard is decentralization—the supervisory rather than the control method; the supervisory method being investigation, comparison, conference and wise publicity. No new building for mental patients should be built without first having its trustees and superintendent appointed in order to plan for its economical medical administration.

A discussion of the internal affairs of such a district (state) hospital is of great interest and brings new ideas. The problem of the dual control from the business and medical sides is attacked by a division of the hospital's functions into four spheres—clinical, scientific, communal and administrative, the latter comprising the nonmedical administration relating to business affairs separate from the care of patients. Each of the spheres so marked must be regarded as coordinate with the others; each should be headed by a director experienced in its special functions, the director of the administrative having technical education and full opportunity for development in a satisfying life career. The field of inter-relationships between all four departments must be left to a medical superintendent, who must act as harmonizer, have final decision subject to appeal to the governing board, but be big enough to give each director full scope in his own department.

The last point in logical order made by Dr. Copp is rehabilitation with a field inside the hospital consisting of systematic training and habit formation and including the occupational homestead and a field outside comprising community psychiatric service.

To one interested in legislation which concerns mental diseases, to any one planning a new hospital, this paper will offer help in big and in little things. It is a sad thing to see money, desperately needed by afflicted human beings, wasted because of ill considered plans.

Bond, Philadelphia.

OCULAR DISTURBANCE IN ENCEPHALITIS LETHARGICA. P. P. Waaidenburg, Am. J. Ophth. **4-8**:580 (Aug.) 1921.

The diagnosis of epidemic encephalitis in many cases cannot be made from the presence of any constant syndrome of clinical symptoms, but the ocular symptoms have proved helpful in fixing the diagnosis in many instances, especially because the disease is characterized not only by the frequency of the well-known isolated or combined nervous paralysis which we meet in other infectious and nervous diseases, but because supranuclear disturbances of the fibers or centers of associated movements often precede the other symptoms.

Three other diseases give differential diagnostic difficulties: (a) nona in which bulbar symptoms are absent, (b) premature paralysis agitans which proceeds more slowly and without inclination to sleep, and (c) bulbar paralysis in which changes of the ocular muscles and pupillary changes are absent. The author elaborates on the cases described by Nonne and Economo and then presents three cases. The ocular symptoms only will be discussed. Ptosis is the most frequent and is more or less fugacious and is usually combined with diplopia; the degree is varied from a paresis to a paralysis. Visual disturbances are due to a retrobulbar neuritis and is manifested by a ring scotoma which is not always isolated but often connected with a central scotoma or enlargement of the blind spot, and it often has inconstant and capricious shapes. Ring scotomas have also been described in multiple sclerosis, hereditary neuritis, syphilis, retrobulbar neuritis, affections of the optic nerve arising from clinical causes, and glaucoma. Paresis of accommodation is also a frequent symptom, but it is also found in syphilis, botulism and diphtheria.

Paresis of convergence is less frequently found. Anisocoria is a cardinal symptom and present in about 20 per cent. of cases. The degree of dilatation is inconstant; the reaction is present but slow, the amplitude slight. The author explains this phenomenon in the following way: "Because the nuclei of the sphincter must be small, the hypothesis is not hazardous that all supranuclear reflex paths which attain this nuclear from about the same direction must lie very close together in some place. If we accept a partial or total destruction of these paths, whether or not accompanied by a slight irritation or paresis of the sphincter nucleus, many facts can be explained in a plausible way; that is, the long duration of the condition, restoration in a different sequence, the different combinations of symptoms, etc. Thus a normal reaction of convergence can be combined with an isolated paralysis of accommodation, and the other combination mentioned."

Paresis of the associated movements, too, are often disturbed and are often combined with total or partial contraction of the homonymous parts of fields of fixation; vertical paralysis predominates over horizontal.

Nystagmus is also a frequent symptom and is due to a disturbance of the vestibular nuclei or the connection fibers between them and the nuclei of the oculomotor or the tonic centers of the conjugated movements.

Concerning localization of nuclei and fibers—we have not advanced much further than the hypothesis: the paralysis of convergence speaks for the existence of a center of convergence placed in the median nucleus of the oculomotors. Besides the oculomotor phase, the facial, which lies at a greater distance, is often attacked, so that we are forced to accept the theory that focii lie more dispersed rather than that they are in continuous connection. We are, therefore, forced to accept an eclectic action for the verus for definite cerebral parts.

DeLong, Philadelphia.

THE CONNECTIONS OF THE VOMERONASAL NERVE, ACCESSORY OLFACTORY BULB AND AMYGDALA IN AMPHIBIA. C. Judson Herrick, J. Comp. Neurol. **33**:213 (Aug. 15) 1921.

The vomeronasal organ, or organ of Jacobson, has long been a puzzle to comparative anatomists and physiologists. Generally present in air-breathing animals, this portion of the nasal organ may contain a considerable part of the specific olfactory membrane, or in microsmatic forms like man it may be vestigeal. When well developed, this portion of the olfactory sensory epithelium has its own nerve, the vomeronasal nerve, which terminates centrally in a special part of the olfactory bulb, the accessory bulb. These structures are absent in fishes and appear in definitive form in the frog and in most reptiles and mammals. The first appearance of this apparatus was apparently correlated with the opening of the posterior nasal aperture, and recent observations of Broman suggest that it is primarily a mouth-smelling organ, that is, a special portion of the nose adapted for smelling odorous mediums within the mouth.

In the frog a special olfactory tract leaves the accessory olfactory bulb to terminate in a differentiated nucleus which is regarded as the precursor of the mammalian nucleus amygdalae. The other connections of the anuran amygdala are: with the opposite amygdala through the anterior commissure, with the adjacent pyriform lobe, with the medial olfactory areas, with the habenula, and with the hypothalamus, the latter by descending and ascending fibers of the olfactory projection tract of Cajal. Through these connections the amygdala of the frog serves as a center for the correlation of odorous excitations derived from the mouth cavity with those derived from the air outside and of both of these with taste and other visceral functions of the hypothalamus.

This appears to be the inception of the amygdaloid complex, which primitively in amphibia was differentiated under the influence of specific sensory excitations coming from the vomeronasal organ. So far as the preexisting nervous pathways connected with the ventrolateral area of the cerebral hemisphere conducted excitations physiologically congruous with those from the vomeronasal organ, these were integrated into a morphologic unity as we see it in the amygdala of the frog. The amygdaloid complex, having been thus integrated under the dynamic influence of vomeronasal excitations, further complication of the system progressed in higher vertebrates in various directions by the incorporation of other related sensory systems, with perhaps profound modification of the functional aspect of the complex as a whole. So great may be this deviation from the primitive physiologic pattern in some cases that the suppression of the vomeronasal organ, as in man, or even of the entire olfactory system, as in the dolphin, does not destroy the integrity of the surviving components of the amygdaloid complex, which retains its individuality in modified form.

Other parts of this paper include a description of the corresponding parts of the brain in urodele amphibians, in which the first stages of this differentiation are recognizable, but without coming to consummation in the complete elaboration of either vomeronasal organ or amygdala. There is also a discussion of the morphogenesis of the strio-amygdaloid complex and a brief review of the comparative anatomy of this apparatus.

AUTHOR'S ABSTRACT.

TECHNIC OF NERVE SURGERY. K. WINFIELD NEY, Ann. Surg. **73**:37, (July) 1921.

The author, having had, within the short span of fourteen months, more than 1,500 peripheral nerve cases under his immediate care, has presented on the basis of this experience a system of procedure and technic in the operative treatment of peripheral nerves, based on physiologic indications. In the suture of mixed nerves, torsion of the nerve trunk results in the physiologic misdirection of nerve fibers, in which motor fibers are directed down sensory channels and sensory fibers down motor channels, resulting in a physiologic loss of function. The author suggests several methods for the prevention of torsion. Nerve defects are considered, and their method of correction is taken up in detail. Improvements in technic have almost entirely eliminated the necessity of grafts for filling defects. Most of the grafts used in his clinic were eventually removed, because of nonregeneration, and it was found possible to overcome most of these defects by the methods described and to accomplish end-to-end suture.

The technic of suture in complete and partial divisions is presented with many illustrations to clarify the text. Physiologic interruptions are classified according to the factors responsible for their production: (1) external compressions due to scar tissue, bone callous, or any form of pressure; (2) thickening and infiltration of the nerve sheath; and (3) interstitial fibrosis (perifunicular).

A definite surgical technic is shown for the correction of each condition. One case of ischemic paralysis of the leg which had existed eighteen months without improvement is reported: blood vessels were exposed and found to be practically normal; the sciatic nerve was opened revealing extensive perifunicular adhesions; the individual bundles were then separated and the sheath left open. There was a return of function in practically all the muscles within three months.

The author lays special stress on the influence of technic on final functional results, in which the amount of scar tissue is diminished by the avoidance of tissue trauma, infection and by careful hemostasis.

In conclusion, he states: "The methods suggested in this paper for the prevention of torsion have given fairly satisfactory results, but now and again we find cases in which in spite of these precautions taken to prevent torsion it has evidently occurred, and there has been a diminished functional return in certain muscles. In some cases a return of function has been so perfect in most of the muscles that it requires a careful examination of individual muscle function to determine motor imperfections, and no doubt much of the resulting disability will be expressed only in a diminished disability to perform certain skilled movements, and there is a possibility that even these minor motor defects may in time be corrected in many individuals by reeducation and substitution movements."

Author's Abstract.

PILOUS VIRILISM AND ITS ASSOCIATION WITH GLYCOLYTIC INSUFFICIENCY. C. Achard and M. J. Thiers, Bull. de l'Acad. de Méd. **85**:51 (July 19) 1921.

The first portion of this paper is both historical and descriptive of hirsutism as it sometimes appears in women. According to Apert, who is quoted, there are three fundamental characters which it possesses, namely, pilosity of a special topography, adiposity, and disorders of the genital functions. One can observe diverse anomalies of the sexual characters, notably mammillary atrophy, hypertrophy of the clitoris or a masculine voice. But all these associated phenomena are inconstant.

We are badly enough informed regarding the mechanism which, in the physiologic state, determines the growth of hair in certain regions in the case of the male adolescent. Its pathologic development in the infant of either sex or in the adult female is even more obscure.

Besides the frequent anomalies in the genital sphere, uterine malformations —such as bicornous, unicornous or double uterus, ovarian cysts, etc.—suprarenal lesions are frequent. It is difficult to define the nature of the genital disturbance which directs this hirsutism. It may be due to an overactivity of the suprarenal cortex. But every suprarenal lesion does not have this result, and one can find among infants suprarenal tumors without coincident premature development.

The writer then gives in detail the history and necropsy findings in a case of hirsutism associated with a glycosuria which was not permanent. The hypertrichosis began at 9 or 10 years of age. The patient had had regular menstruation which lasted to the unusual age of 60, and she had had one pregnancy. At the age of 69, following a severe trauma, she showed a glycosuria which disappeared under mild regulations of diet. He contrasts this case of hirsutism associated with glycosuria with others of recent reporting (Emile-Weil and Plichet; Tuffier and Guemes, Laignel-Lavastine and Boulet). Regarding their menstrual or reproductive functioning, there is no uniformity in the series whatsoever. Also the anatomic variations in the genital sphere are as variable as in so-called simple hirsutism. Again, the suprarenal capsules in some, but not in all, instances have presented lesions.

He calls attention to an increase in the cholesterin content of the blood in these cases. Finally, the hypertrichosis made its appearance at very variable dates—in the case reported before the onset of menstruation, at the menstrual epoch in another, between two pregnancies in another and at the menopause in one case.

The degree or severity of the "diabetes" associated with hirsutism is also extremely varied, as the author shows by citing figures from the assembled cases, and he considers that glycolytic insufficiency is only an inconstant and variable element in feminine hirsutism of the virile type. He takes its occasional appearance as proof of the participation of several organs in a pluriglandular syndrome.

Davis, New York.

NEUROTROPIC FILTRABLE VIRUS. Paul Harvier, Paris méd. **2**:485, 1921.

This interesting review is summed up as follows:

"Epidemic hydrophobia, poliomyelitis and encephalitis belong to the same group of infectious diseases, brought on by filterable virus having an elective affinity for the nervous system."

There are analogies and differences between these three diseases which are summed up thus:

These viruses exist in the general nervous system in a pure state. These diseases can be experimentally reproduced and transmitted from animal to animal by inoculation of an emulsion of the central nervous system or by the filtrate of that emulsion. The inoculation gives positive results, not only in the intracerebral path, but also in the ocular path, the peripheral nerve path and nasal path (after scarification or traumatism of the membrane).

The virus of encephalitis, alone, produces a specific keratitis. The virus ascends the nerves to reach the cerebrospinal axis where it grows during the period of incubation of the disease.

These different viruses act in a fashion analogous to physical and chemical agents. They resist light and desiccation; they are killed by heat of 55 degrees and by a certain number of antiseptics; they conserve their virulence in glycerin. They are destroyed by bile.

According to American bacteriologists, their culture in Noguchi mediums shows bacterial forms very closely related.

The inoculation of these cultures produces the experimental disease. The parasite may be determined in the central nervous system of those animals which succumb to the disease.

The relations of epidemic poliomyelitis and encephalitis are more intimate than those between these two and hydrophobia. The first two diseases are transmitted by the same pathway—the nasopharynx. The presence of their virus was shown in the nasal membrane and the nasopharyngeal secretions. The propagation of these diseases seems to be accomplished by the sick, the convalescing, perhaps the healthy persons living with or near these sick people and by "virus carriers."

However, besides the analogies, fundamental differences exist. These consist, above all, in the unequal receptivity of the various species of animals with regard to each of these viruses, in the contents of the serum of vaccinated animals and of cured cases, and in the particularities of immunity.

The serum of animals vaccinated against hydrophobia neutralizes in vitro and in vivo the virus of hydrophobia. Similarly, the serum of animals cured of an attack of poliomyelitis neutralizes the virus of that disease. Epidemic encephalitis acts differently. The attempts at vaccination, of neutralization of the virus by serums, has given, up to the present time, only practically negative results.

A cross immunity between hydrophobia, encephalitis and poliomyelitis does not exist. Animals which have natural immunity to the virus of poliomyelitis are susceptible to hydrophobia.

The virus of encephalitis does not vaccinate the monkey against poliomyelitis. In a like manner, the virus of poliomyelitis does not vaccinate the rabbit against encephalitis.

(This article was extracted by P. H. from "Infections à Virus Filtrants" from "Nouveau Traité de Medecine at the Thérapeutique" published under the supervision of M. Gilbert and Carnot, vol. 10 [in preparation].)

Kraus, New York.

ON THE BREAKING OF FAMILY—AND OTHER—TIES. Kraepelin, Ztschr. f. d. ges. Neurol. u. Psychiat., O. **63**:1-8, 1921.

Americans will find a contribution by Professor Kraepelin interesting, even if it seems rather remote in its way of dealing with its subject. This subject, the effects on a man of cutting him off from the different circles to which he was born, seems too big to be handled when it involves human relationships from family to nation.

Man as a social being is discussed. The author considers that, certain essentials being fixed by heredity, the development and direction of a person is largely controlled by those personal contacts which are most powerful in youth. Given some weakness of character to start with, the child and youth turns to the family for protection and help in trouble, and he turns with a sense of "belonging." It is the family that has the best chance to smooth his rough places, laugh him out of his conceits, and teach him the beneficial results of easy communication with his fellows.

Ordinarily when the child turning for help finds step-parents, he suffers. Something is gone for which the kindest treatment is not a substitute. He is one step removed from "his own people."

The effects on the personality will evidently vary according to the causes of the separation from the family or nation. On one side great external accidents such as death may do the disrupting; on the other side the personal peculiarities of a member of the family or oneself. There follows a rather formal account of the possible variations of separation in degree and in kind. It may be complete or incomplete. The illegitimate are affected in one way and the orphans in another; the bearer of a great name and a person marked by the commonplace and by poverty; the city merchant and the peasant. A father drunk may produce the effect of a father dead. There is a different problem with the only child and with the child surrounded by brothers and sisters. And the effect must be graded according to age; it is doubtful whether it is stronger in childhood or in puberty; at a certain age it is normal.

The evil tendency consequent on breaking natural ties at the wrong time is seclusiveness and self-investigation. The means with which to fight it is an expansion of social psychiatry. Schools must make a scientific study of the problem. States must study colonization. More attention must be paid in general to the effects on character of leaving, or being left by, one's own family, neighbors or nation.

Bond, Philadelphia.

PARKINSONIAN SYNDROMES. Fernand Levy, Gaz. d. Hôp. **94**:757 (June) 1921.

This is a review of the paper by Souques and the discussion, presented at the Paris Neurological Society in June, 1921. The meeting was devoted to the parkinsonian syndrome.

Introduction.—This syndrome is defined as follows: "Paralysis agitans is not a morbid entity, but a syndrome resultant on various causes acting on the same part of the brain."

Pathologic Anatomy.—The following theories of the location of the lesions were given:

(a) Ramsay Hunt: A lesion of the globus pallidus of the striate body. This lesion affects the pallidal system.

(b) Tretiakoff: This author believes the lesion is due to atrophy and degeneration of the cells of the substantia nigra.

(c) S. A. K. Wilson emphasizes lesions of the red nucleus.

(d) These theories are not at variance fundamentally according to Mirto, who regards the substantia nigra as a part of the globus pallidus, separated from it in the course of phylogenetic development.

(e) Foix believes that the lesion is a diffuse one of the striate body not limited to the globus pallidus.

The types of lesions include tumors, cerebral hemorrhage, cerebral softening, infections, lesions, toxic processes, etc. These lesions must, in order to produce the syndrome, affect either the striate body or the substantia nigra —which of these must be decided in the future. A more or less extensive involvement of the centers of tonus results (Souques).

Etiology.—(a) The syndrome cannot longer be considered a neurosis. Fear may play a part in precipitating the disease. It does not cause it but may, acting on suitable soil, bring it to light.

(b) Trauma is to be considered as a precipitating factor in a few cases. Souques, who saw a great many war cases, both of emotional and concussional shock, never saw paralysis agitans as a sequel.

(c) Infections are now the best established causes of the syndrome. Before the epidemic of encephalitis this was not generally recognized, though cases following scarlet fever, typhoid, measles, articular rheumatism, malaria and syphilis were reported. Epidemic encephalitis is the most frequent cause.

The syndrome comes on, as a rule, between the ages of 40 and 60, though it may occur in youth and childhood (3 years).

Symptoms.—The main symptoms are rigidity, tremor and loss of associated movements.

These symptoms are described in too much detail for review. Pain is briefly considered. It is present in about one sixth of the cases. Vasomotor disorders are not infrequent. The rest of the review is devoted to a section on clinical forms, diagnosis and treatment.

The clinical forms described are the classical type, a forme fruste with rigidity and no tremor and a forme fruste with tremor and no rigidity.

No essential difference exists, according to Souques, between the juvenile and presenile forms. Neither the section on diagnosis nor that on treatment contains information that is not familiar.

Kraus, New York.

THE COURSE OF PARESIS. Friedrich Meggendorfer, Ztschr. f. d. ges. Neurol. u. Psychiat. **63**:9, 1921.

The author reports on the findings in over 1,200 cases of general paralysis of the insane. Concerning the incubation period, the author shows graphically the latent period between the syphilitic infection and the onset of the paralytic symptoms. With an infection in youth the latent period is long, paresis rarely setting in before the thirty-fifth year, twenty years after the initial lesion. With increasing age at the time of infection the incubation period of paresis grows shorter, until with an infection coming first at 50, a latent period of barely five years may be expected before the onset of the paralysis. Moreover, with a late onset the course is apt to be shorter and more stormy and lead to an earlier death. Cases of juvenile paresis due to congenital syphilis are, of course, excluded.

According to the author, and as is frequenty observed, paresis develops in cases of syphilis with the milder secondary manifestations; moreover, the treatment of syphilis by mercury and iodids seems to have a shortening rather than a lengthening effect on the latent period before the onset of paresis.

The infrequency of paresis in prostitutes is accounted for by the early age at which most of them are infected with syphilis, the prolonged latent period permitting many other opportunities for degeneration and death before the paresis has become established.

The tendency of paresis to occur during the fifth decade of life is strongly emphasized by the author, and in explaining this occurrence it is noted that this is the decade of involution, when the endocrine tissues are undergoing degenerative processes, the internal chemical processes are less vigorous, and the forces of resistance of the body are being broken down.

Finally, the much vexed question of the elective action of certain "strains" of the spirochete for the nervous system is taken up. Meggendorfer theorizes thus: A person, owing to disease or injury of the nervous system, becomes more susceptible to injury at this point. He becomes infected with syphilis, and the spirochete attacks the weakest tissue. He already has the "disposition to metalues." He develops paresis, and infects another with syphilis. The spirochete, causing a mild general infection, will localize in the central nervous system and produce paresis again. Thus the strain will be determined. In support of this theory he brings in the observation of Pilcz, that the Hungarians are prone to develop paresis, but that the person of foreign race, contracting syphilis on Hungarian soil, will be far more apt to develop paresis after infection with the spirochete, than he will be if infected in any other part of the world.

Freeman, Philadelphia.

CONVULSIONS IN EARLY INFANCY. A.-B. Marfan, Presse méd. **29**:633 (Aug. 10) 1921.

Convulsions of all types in this age-period are almost always accompanied by loss of consciousness. They tend to be repeated. The age itself is the greatest predisposing factor; most infantile convulsions occur in the first two years of life, particularly from the third to the sixth month.

The "complete, general, and regular" convulsion resembles that of ordinary epilepsy in its successive stages. The onset is oftenest instantaneous, the duration generally very short (a few seconds to a minute or two). Status is not rare. Atypical tonic-clonic convulsions are more common than the typical in this age-period. They may be incomplete (tonic spasms without clonic, or clonic without apparent tonic), partial (usually affecting some portion of the face) or irregular (tonic and clonic spasms alternating variably).

Precipitating causes of infantile convulsions may be: (1) organic, almost any acute or chronic central nervous lesion; (2) symptomatic, an affection of another system, as in infections, intoxications, and sudden pyrexias from any cause; (3) a spasmophilic diathesis, with tetany (Marfan disputes Escherich's assertion that all cases with early infantile convulsions show signs of latent tetany between attacks); or (4) idiopathic.

Group 4 is losing in favor of Group 1 through more intensive and successful searching for signs of heredosyphilis. In the few residual cases that must still be called idiopathic, epilepsy is not necessarily to be expected as a sequel.

The question whether a convulsion is taking place sometimes arises with laryngospasm (a localization of tetany), which may or may not precipitate a true convulsion. Spasmus nutans may also be confusing.

In any type of infantile convulsion, the immediate and remote prognoses are both more grave if the tonic spasm is unusually marked or prolonged.

When phenylethylmalonylurea is used, Marfan recommends a large dose of ⅓ to ½ grain daily for infants under 6 months. He also states that boric potassium tartrate is badly tolerated.

Huddleson, New York.

ACUTE INFLAMMATORY TABETIC ARTHROPATHIES. A. Lemierre, M. Léon-Kindberg and P. N. Deschamps, Gaz. d. hôp. **94**:901 (July) 1921.

These authors describe a case of acute inflammation of the left knee which proved to be of tabetic origin. After the patient had remained in the hospital a week a backward luxation of the tibia occurred. The knee and Achilles reflexes were absent on the unaffected side. The spinal fluid showed lymphocytosis.

The arthropathy was characterized by severe pain, frank local signs of inflammation and a general febrile reaction.

The authors comment on the relative rarity of the condition, the severity of the pain, the diffuseness of the swelling (from the ankle upward in their case), the suggestion of erysipelas in some cases, the transitory general febrile reaction and the bad prognosis. The prognosis is bad not only as to the local condition, but often also as to the general condition of the patient. After the febrile attack, the disease progresses rapidly and often ends in death. In some cases, however, this does not occur and the patient, after recovering from the acute attack, presents chronically, the classical picture of a tabetic arthropathy. Secondary purulent infection may occur.

Treatment by antisyphilitic drugs is not effective, since, as these authors point out, the damage has already been done. They suggest surgical interference in some cases, opening of the joint, and curetting of the necrosed tissues for the purpose of eliminating a dangerous focus of infection.

An interesting account of the pathology of these acute tabetic arthopathies is given. This emphasizes the syphilitic etiology and the difference of the findings from those of coccal forms of arthritis. The paper is interesting and thorough.

Kraus, New York.

THE VASCULARITY OF THE CEREBRAL CORTEX OF THE ALBINO RAT. Edward Horne Craigie, J. Comp. Neurol. **33**:193, (Aug. 15) 1921.

In a previous paper the author described a standardized method for determining the relative vascularity of parts of the central nervous system and presented quantitative data on specific regions of the spinal cord, brain stem and cerebellum of the albino rat.

The vascularity of the cerebral cortex is of about the same order of magnitude as that of the cerebellar cortex. The vascularity of each of the five recognizable layers was measured in five different areas of the cerebral cortex. In all areas the lamina granularis interna (IV of Brodmann) is more richly vascularized than are the other layers, and the supragranular layers tend to be richer than the infragranular layers, a result of considerable interest in connection with current beliefs that the supragranular layers serve higher cortical functions than the infragranular layers. The author believes that the granular and supragranular layers are receptive and associative in function, while the infragranular layers give rise to corticofugal fibers. This suggests a comparison with the lower centers, where the sensory and correlation nuclei were found to be more richly vascular than the motor nuclei. The average vascularity of the five layers is the same in the occipital and temporal areas, and is only slightly less in the precentral region. The parietal area is distinctly richer than the others, while the insular cortex is much the poorest.

C. J. Herrick, Chicago.

THE PSYCHOPATHIC PRODROMAL SYMPTOMS OF EPIDEMIC ENCEPHALITIS. P. Chavigny and E. Gelma, Bull. de l'Acad. de méd. **85**:113 (July 26) 1921.

Since the work of Netter, Achard, Legre and Bremer, the frequency and variety of psychic symptoms in the prodromal period of epidemic encephalitis have been recognized. These symptoms are characterized by mental confusion, and behavior on an impulsive basis sometimes transitory and followed by post-confusional lacunar amnesia, and they appear before the onset of the commonly recognized usual symptoms of the disease. If a patient in the prodromal period, commits an unlawful act leading to arrest, and if a short time after his arrest, the confusion has disappeared, one can easily fail to understand the morbid nature of the felonious action and may believe that the subject is simulating an amnesia.

A young peasant became ill with fever, photophobia, frontal headache, stiffness of the neck, diplopia and spinal fluid findings in accord with the diagnosis of encephalitis, on the day following his arrest for the theft of a bicycle. He was in a confused state, was imperfectly conscious of his situation and was unable to recall the reason for his incarceration. The writers present the amnesia encountered in the case as legitimate and relate that he was declared irresponsible by reason of his state of consciousness at the time of committing the act. Psychiatrically there was not alone a post-confusional amnesia but confabulation with a mingling of true and false memories, of post-delirious ideas and fantastic interpretations. He remained unable to relate himself to the reality of the past in spite of reiterated explanations made before him.

A possible hypothesis of simulation may not be sustained for several reasons —the certitude of the clinical encephalitis which arose only a day following the theft, the knowledge that disordered and extravagant acts are sometimes features of its prodromal psychic symptomatology, and the conviction that the correctness of the clinical psychiatry pictured, confusion, amnesia and confabulation was beyond the simulating powers of a peasant.

Davis, New York.

INTERNAL SECRETIONS AND THE NERVOUS SYSTEM. W. Löffler, Schweizer Arch. f. Neurol. u. Psych. **8**:163-183, 1921.

It is extremely difficult to formulate any theory explaining the physiologic and pathologic processes going on in the organism that does not include the conception of internal secretions. The doctrine of internal secretions, however, has developed into a form of mythology in which the various hormones are enthroned and deposed, very much like the gods and demigods of the ancients, as the necessity of the writer demands.

The correlation taking place between various organs in the body may proceed according to nervous, chemical and neurochemical means. These distinctions, however, cannot always be clearly drawn. For example, as a purely chemical correlation may be mentioned the production of urea in the liver. An example of a neurochemical process would be the classical experiment of puncture diabetes.

The writer reviews the theories relating to various organs of internal secretion, such as the hypophysis, the epiphysis and the hypothalamic region and their relation to diabetes insipidus, Raynaud's disease and other angiospastic conditions, and dystrophia adiposogenitalis. The relation of the epiphysis to macrogenitosomia praecox and the circulation of spinal fluid in the aqueduct is briefly considered. The choroid plexus, the chromaffin system, the thyroid and finally the testicles are considered in turn.

In all of these Löffler pictures the process by which this metabolic correlation is brought about as being dependent on a special arrangement of substances within the cell, changes in surface tension and alterations of osmotic pressures. In this way chemical substances can be separated within the cell itself so that reactions between them become impossible unless the factors enumerated permit it. Sympathicotonia and vagotonia are dismissed by a brief reference to the difficulty encountered in attempting to set up sharply defined clinical pictures.

Altogether, Löffler's article gives a satisfactory, although very brief, review of this intricate subject, without the addition of any original contribution.

Woltman, Rochester, Minn.

PSYCHOLOGICAL TRAITS OF THE SOUTHERN NEGRO WITH OBSERVATIONS AS TO SOME OF HIS PSYCHOSES. W. M. Bevis, Am. J. Psychiat. **1**:69 (July) 1921.

Bevis presents a psychologic study of the Southern negro. His general conclusions, such as the fact that motion, rhythm, music and excitement make up a large part of the life of the race, that as a people they are carefree and improvident and unable to profit by experience and that fears and superstitions are almost universal among them perhaps merely reflect intellectual immaturity. The negro responds to his environment by a group of relatively simple affective reactions. It has been noted that the formation of a neurosis in the negro usually does not involve a complicated mechanism.

The incidence of cerebrospinal syphilis and paresis was found to be relatively low in the Southern negro. This observation has been made frequently and is by no means peculiar to the negro. Kraepelin originally called attention to the practical immunity not only of the negro, but also of the North American Indian in his natural state, the native Algerian and others. Perhaps this and likewise the surprisingly low percentage of alcoholic psychoses may be interpreted as indicating not only elemental psychologic traits, but anatomically a "lower plane" central nervous system, that is, a lack of complexity and finer differentiation.

Additional statements of interest are that the manic phase of manic-depressive patients is much more usual than the depressed and that dementia praecox is the common psychosis. This is especially true of the catatonic form.

Strecker, Philadelphia.

NEUROSES IN BUSINESS LIFE. Louis Casamajor, Neurol. Bull. **3**: 237, (July) 1921.

Casamajor discusses in a broad way some of the neuroses in industrial life and finds in them the same underlying causes as in the war neuroses. He urges that the idea of traumatic causation be discarded and that study be directed to the personality and psychologic adaptation of the individual. It is his opinion that "workmen's compensation laws . . . for the psychoneuroses have been but stimuli for the development and persistence of sickness"; nevertheless, he feels that desire for compensation alone, which is a conscious process and is malingering, is untrue for most cases. He therefore stresses the unconscious motives in the neuroses of business life as in all other psychoneuroses.

Roughly he groups his cases into: (1) inadequate; (2) dissatisfied, and (3) generally maladjusted. Characteristic of all is the dissatisfaction with the job and the more or less unconscious reason for the conflict.

1. The inadequate person is unequal to his job, fears that he won't succeed and develops his symptoms when responsibility is thrown on him. The result is usually an anxiety neurosis, and his sickness (unconsciously) justifies his failure and saves his self-esteem.

2. The dissatisfied form the largest group. Although equal to the task, these people don't like the work itself, wherefore any trauma is apt to serve as an excuse for giving it up. Most "normals" more or less belong in this group, without, however, crossing the borderline into a true neurosis. Patients of this stamp are easily cured with analysis or suggestion.

3. The generally maladjusted in business life exhibit but a part of their maladjustment to life in general. The latter is really their greater problem. Some of these patients overwork themselves to overcome life's difficulties and often crack as an unconscious way of getting out of them. A slight trauma may be sufficient to cause a litigation neurosis.

Wechsler, New York.

ON AN ENDOCRANIAL CAST FROM A REPTILE, DESMATOSUCHUS SPURENSIS, FROM THE UPPER TRIASSIC OF WESTERN TEXAS. E. C. Case, J. Comp. Neurol. **33**:133-147 (June 15) 1921.

The determination of the exact relations between a cast of the cranial cavity and the brain originally occupying that cavity is a matter of great anthropologic significance. In the study of prehistoric remains, whether of men or other vertebrates, it is necessary to bear in mind that the process of fossilization may yield endocranial casts, endodural casts, or (rarely) actual brain form. These must be carefully distinguished.

In this contribution to paleoneurology the author describes a new endocranial cast of a Triassic reptile and copies from the literature figures of similar casts from several other extinct reptiles. He comments on the fact that in some of these gigantic reptiles the pituitary fossa was very large and says, further: "In all of these forms the brain is exceedingly small relative to the body, but the pituitary body is very large relative to the size of the brain. The connection between the size and activity of the pituitary gland and the size and the proportions of the body in mammals is well known. Hyperpituitarism results in large size or the overdevelopment of certain structures, as the fingers, features, etc. It is equally well known to paleontologists that one of the common variations among those which occur so abundantly in the senile stages of any phylum is giantism. The suggestion naturally arises that the disturbances which arose in the phylum coincident with the development of adverse conditions were primarily of a physiologic character affecting the deep-seated organs of the nervous system and through them, and only secondarily, the superficial structures. The teratologic affections of the pituitary body which produced abnormal structures in a normal phylum may have gradually become a fixed character and resulted in normal giantism in certain groups."

C. J. Herrick, Chicago.

REPAIR OF CRANIAL DEFECTS BY BONE TRANSPLANTS. A COMPARISON WITH CARTILAGINOUS TRANSPLANTS AND ENDOPROSTHESIS. Mauclaire, Paris méd. **2**:153, 1921.

The author reviews the subject beginning with a classification of transplants. His review is an extract of a book, "Etudes sur les Greffes chez l'homme et chez les animaux" (J. B. Ballière et fils), to appear shortly. He divides transplants into two large groups, those of living bone and those of dead bone.

The indication for transplanting is a defect. Contraindications are increased intracranial pressure, underlying chronic meningitis and a defect larger than 6 by 6 cm.

He discusses the varieties of transplants at length and concludes that the results of bone transplantation are good when no cerebral or meningeal complications are present. In such cases the symptoms of the post concussional neurosis remain to some degree. He cites Pièrre Marie's figures on twenty-one cases in which six were improved, twelve remained the same as before filling in of the defect and three were worse. He cites the cases of Henri Claude and Sicard in which the patients were so much worse that the transplant had to be removed.

He ends by saying that the transplants do not remove symptoms of central origin.

Kraus, New York.

ATYPICAL FORMS OF EPIDEMIC ENCEPHALITIS. Charles Rosenheck and Leon H. Cornwall, Neurol. Bull. **3**:259 (July) 1921.

The authors describe six cases with mild signs which they would include in the epidemic forms of encephalitis. One patient gave signs of a mild transverse myelitis, two showed pupillary signs suggestive of syphilis, all had pains which were radicular or neuralgic in character, a few that manifested distinct depressive and psychoneurotic symptoms, and nearly all had unequal reflexes and hyperesthetic areas. Ordinarily, the authors assert, no diagnosis of encephalitis (or rather meningo-radiculo-myelo-encephalitis) would have been made. A spinal puncture, however, in each case, confirmed their suspicion that they were dealing with atypical forms of the disease. The punctures showed increase of cells, increase of globulin and increased sugar reduction. Negative Wassermann and colloidal gold reactions served to rule out syphilis. They stress the importance of sugar reduction.

It is quite probable that the cases described belong to the epidemic variety, but exception may be taken to the term atypical. Such cases have occurred and the word mild more aptly describes them. Some have undoubtedly been overlooked and more will be missed in the future as the memory of the epidemic slowly fades. The spinal fluid findings do, of course (although not always), help, but one is not always ready to perform puncture on ambulatory patients. It may be said, too, that for every mild, abortive, atypical or ambulatory case that has been overlooked another has been erroneously diagnosed as encephalitis. However, although every neurologist has encountered such cases, the authors have done well to call attention to their occurrence at this time.

Wechsler, New York.

EPIDEMIC ENCEPHALITIS OF LONG DURATION. S. Turretti and G. Piotrowski, Paris méd. **2**:348, 1921.

These authors have found but four cases in the literature of long standing epidemic encephalitis.

The first case recorded by these authors is that of a girl, aged 24, whose illness began in February, 1920, following an attack of "grippe." She suffered from choreiform movements, insomnia, headache and a moderate rise of temperature at the onset. The movements soon ceased. The insomnia at night and somnolence during the day increased. The patient had to be tube-fed. The following September, eight months later, the patient was still very sick, though her condition had improved. She showed marked emaciation. Rigidity of facies and body was still present. Mentally, she was clearer. Her memory for past events was intact.

The second case is that of a man, aged 45, who was taken sick in April, 1917. He presented many of the classical symptoms of epidemic encephalitis. He died in January, 1919, having been sick twenty months. At necropsy, evidence of recent and old encephalitic lesions was found.

The authors believe that these prolonged cases occur in patients showing lethargy, and that the lesion lies in the striate body. They emphasize this by recalling how much the symptomatology resembles that of progressive lenticular degeneration described by S. A. K. Wilson.

This abstract may give the impression that prolonged cases of epidemic encephalitis are rare. This is not the fact, for many can be found in most clinics. The possible duration of the disease still remains to be settled.

Kraus, New York.

PROGRESSIVE MUSCULAR DYSTROPHY AND THYROID HYPOPLASIA. Hellmut Maiweg, Ztschr. f. d. Ges. Neurol. u. Psychiat. **63**: 107, 1921.

In reporting a case of pseudohypertrophic muscular dystrophy complicated by hypothyroidism, Maiweg calls attention to the frequency with which the former disease is complicated by various other abnormalities, especially of internal secretory functions. Whereas the combination with hypothyroidism has not been described before, the dystrophy has been noted in cases in which there were infantilism, skeletal anomalies, disturbances of intellectual development, exophthalmos, hypertrophy of the tongue, penis, thymus and mammae, hyposexuality and the Chvostek phenomena of tetany. The author lays special emphasis on the disturbance of function of the thyroid and parathyroid glands, and suggests that a more careful examination may reveal hypothyroidism in a greater number of dystrophic cases. In closing he says: "One may look in the future for the consideration of Erb's dystrophy among the disturbances of the internal secretions, especially under hypothyroidism. Its more frequent determination would be of interest not only from the symptomatic and pathogenetic standpoint, but also from the therapeutic standpoint."

Freeman, Philadelphia.

MYOCLONIA OR INTENTION SPASM MUCH IMPROVED BY THE ACTION OF LUMINAL. THE ACTION OF LUMINAL IN PARKINSON'S DISEASE. Clovis Vincent and Krebs, L'Encephale **16**:260, 1921; Proceedings of the Society of Neurology of Paris, April 7, 1921.

The patient was a young girl of 19 who had had myoclonia of the left arm for three years. The cause of this myoclonia could not be ascertained. The onset had been sudden. The movements were so severe and fatiguing that the patient had to go to bed at 2 p. m.

These movements were almost completely stopped by 0.15 gm. of luminal taken in the morning by mouth over a period of over thirty days. After the luminal was stopped, the movements did not return at once, but slowly and progressively.

In certain patients with Parkinson's disease, neither able to walk nor write, luminal relieved the spasm of the extremities sufficiently to permit both walking and writing. In certain cases, scopolamin in small doses was given with the luminal with better results. Great care, however, must be taken in the administration of this drug.

All cases of Parkinson's disease are not favorably influenced by luminal.

Kraus, New York.

TRAUMATIC LESIONS OF THE PITUITARY BODY, AND MULTIPLE CRANIAL NERVE PARALYSES. L. Reverchon, G. Worms and Rouquier, Presse méd. **29**:741 (Sept. 17) 1921.

This detailed and interesting case history, with two radiograms and three other illustrations, includes the findings at necropsy after several months' observation. Numerous authorities are quoted and compared in reference to each feature of the case.

A line of fracture passed through the posterior part of the sella turcica. The fifth, sixth and seventh nerves were bilaterally affected, and there was general clinical evidence of a hypopituitary condition—especially polydipsia, polyuria, severe anemia, asthenia and arterial hypotension with mental infantilism and lethargy. At necropsy the hypophysis was found reduced to a mere fibrous shred without a trace of normal tissue to be seen under the microscope.

Another traumatic case, reported previously by the same authors, is contrasted with this; the other patient developed tumor of the hypophysis and a different clinical picture.

Huddleson, New York.

OCULAR MANIFESTATIONS IN ENCEPHALITIS LETHARGICA.

Gustavus I. Hogue, Am. J. Ophth. **4-8**:592 (Aug. 21) 1921.

The ocular symptoms are frequently the first to be complained of; the order of frequency is diplopia, ptosis, impaired accommodation, optic neuritis and papilledema.

The diplopia is due to motor paresis or paralysis, is of central origin and usually transient. The nerves involved in order of frequency are the sixth, third and rarely the fourth. Ptosis is usually bilateral, usually partial and due to weakness of the levators. Ptosis combined with ocular paresis is pathognomonic of epidemic encephalitis.

Impaired accommodation is manifested by quick fatigue. This symptom is the last to leave; the pupils are sluggish, irregular, unequal bilaterally, and nystagmus may be slight or pronounced. There is no lagophthalmus.

Optic neuritis is manifested by blurring of the disk and is probably due to dilation of capillaries with resultant congestion of the retinal veins.

Papilledema is a rare condition—there are only two cases on record—and when present is due to subcortical hemorrhage or edema cerebri or both. Four cases are reported.

DeLong, Philadelphia.

EPIDEMIC ENCEPHALITIS AND PREGNANCY. TRANSMISSION OF THE DISEASE BY THE MOTHER TO THE FETUS.

Ricardo Jorge, Paris méd. **2**:454, 1921.

The reported results gathered by Jorge of epidemic encephalitis in pregnant women are grouped as follows:

1. The encephalitis was cured, and the pregnancy was normal.
2. The encephalitis ended in death before delivery.
3. The encephalitis ended in death after spontaneous delivery.
4. Delivery was artificial in order to save the patient.

Of the twenty-five cases reported, the fetus was still-born in three. In sixteen cases no record was made, owing to the death of the mother before the end of pregnancy. In five cases (20 per cent.) the fetus lived. Two of these showed typical myoclonic movements. In one, these stopped in five days. No notes of the others are present. One patient died a few days after birth, and at necropsy showed signs of encephalitis.

The author concludes that congenital epidemic encephalitis exists.

Kraus, New York.

DIPLOMYELIA.

F. Kino, Ztschr. f. d. Ges. Neurol. u. Psychiat. **65**:272, 1921.

Kino's patient was a man, aged 36, who died eighteen months after an apoplectic stroke. The spinal cord was found duplicated from the third to the fifth lumbar segments. Diagrams of the cord at different levels are given by the author, showing how the doubling of the cord resulted from incomplete fusion of the two sides above and below, with the development of two morphologically well formed spinal cords between the levels named. The author distinguishes several differences, however. The most important is a complete absence of any pyramidal degeneration in the accessory cord. In addition, whereas the cells of the dorsal horns are well formed, those of the ventral horns are few in number and poorly developed.

The author suggests that embryologically there was an imperfect fusion of the two sides of the neural fold.

He comments on the relative longevity of his patient and of the other subjects reported in the literature.

Freeman, Philadelphia.

NERVE TERMINATIONS IN THE LUNG OF THE RABBIT. O. Larsell, J. Comp. Neurol. **33**:105-131 (June 15) 1921.

Our knowledge of both the anatomy and the physiology of the intrinsic innervation of the lung is very meager. In this study, sensory and motor nerve endings of the rabbit were investigated by the methylene blue method. Three types of sensory endings were found in the epithelium of the primary bronchi and at the points of division of the succeeding orders of bronchi. The most distal endings were observed in the walls of the atria. These are adapted to respond to stretching of the walls of the atria during inspiration, and may represent the receptors responsible for the inhibition of inspiratory movement and the excitation of expiration. The nerve endings in the larger bronchi are regarded as tactile in function, responding to the presence of foreign bodies; those in the smaller passages may be stimulated by pressure during expiration. Motor endings were found both in the smooth muscle of the bronchial tree and in the muscular coat of the pulmonary vessels.

C. J. Herrick, Chicago.

THE PLATELET COUNT AND BLEEDING TIME IN CATATONIC DEMENTIA PRAECOX. Schichi Uyematsu, Am. J. Psychiat. **1**:15 (July) 1921.

Uyematsu finds that the bleeding time is decidedly lessened in catatonic dementia praecox, the average being 3.3 minutes, while the blood platelet count is markedly increased. The same relationship between the coagulation period and the number of platelets has been described in hypothyroid states, notably myxedema and cretinism. The author's study was stimulated by the clinical similarity existing between clearly recognized and relatively well understood minus thyroid conditions and dementia praecox, which is still a closed book. The attempt to read its pages by the light of endocrinology is fascinating, but still barren of result. Uyematsu deserves credit for his careful investigation and the presentation of a not unimportant point. However, even his rather conservative conclusion in favor of an endocrine origin for catatonic dementia praecox is somewhat in excess of the premises.

Strecker, Philadelphia.

EFFECTS OF CUTTING THE LINGUAL NERVE OF THE DOG. J. M. D. Olmsted, J. Comp. Neurol. **33**:149-154 (June 15) 1921.

Taste buds are known to disappear from the tongue after severance of their nerve supply, but there are differences of opinion as to the manner of this disappearance, whether by degenerative destruction of their cells or by dedifferentiation and return to the form of indifferent epithelial cells. On experimentally cutting one lingual nerve of the dog, the taste buds supplied by this nerve disappear within fifteen days. The process is a true degeneration with the aid of phagocytic leukocytes, not a dedifferentiation or metamorphosis of the cells.

C. J. Herrick, Chicago.

DIAGNOSIS OF THE LOCATION AND NATURE OF A CERTAIN VARIETY OF CEREBRAL TUMORS (PSAMMOMA OR ANGIOLITHIC SARCOMA) BY ROENTGEN RAY. Souques, L'Encephale **16**:259, 1921; Society of Neurology of Paris, April 7, 1921.

Roentgen-ray examination showed with great clearness and precision not only the place, but also the form, volume and relations of the tumor in question. The patient presented signs of tumor. The tumor was removed and the patient was cured. Histologic examination showed that the tumor was a psammoma. The author emphasizes the importance of the roentgen ray in detecting this kind of tumor since it is benign and easily removed.

Kraus, New York.

Society Transactions

NEW YORK NEUROLOGICAL SOCIETY

Three Hundred and Ninetieth Regular Meeting, Oct. 4, 1921
Foster Kennedy, M.D., *President, in the Chair*

A CASE OF ACUTE INFECTION OF THE SPINAL ROOTS AND PERIPHERAL NERVES (NEURONITIS). Dr. Thomas K. Davis.

Dr. Davis presented the patient, a man 20 years of age, who had been a marine fireman. His family history was negative. He had been a hard worker and had enjoyed good health. He had used neither alcohol nor drugs and had had no venereal infection. A year ago, in the South, he had had an illness, probably typhoid, for two months. Several months after recovery, he noticed a weakness in the arms, which was not very disabling. This continued for two months when weakness began in the left leg, progressed and became quite severe. Soon it extended to the right leg, so that in the third month of these symptoms he was unable to walk except with assistance. Bladder control was good; there were no eye symptoms and no somnolence; no signs of epidemic encephalitis.

When first examined, one month ago, he had nystagmus to the left and sometimes to the right. Otherwise the eyes were normal, as were swallowing and speech. There was weakness of the flexors of each arm and of the left triceps. The muscles of the trunk were normal. There was weakness above and below the knees and complete foot drop. Both knee jerks and ankle jerks, and in the left arm biceps and triceps jerks, were absent. The right abdominal reflexes were diminished, the left absent.

He had a left tactile hemianesthesia, not total; on the right he had a stocking type of anesthesia. The hemianesthesia stopped very distinctly at the midline and above at a line with the angle of the jaw. Sensation in the fingers and toes was normal. There was some atrophy of the flexors of the arms, considerable in the legs, but no tenderness on pressure along the nerves. The electrical responses showed very slight variations from the normal.

His general health was good, and we found no evidence of disease of the internal organs. The spinal fluid Wassermann reaction was negative. The spinal fluid had 11 cells per c.c.; the blood pressure was 110; the blood count normal.

He received massage and galvanism and has shown improvement. The abdominal reflexes are now stronger. The knee jerk can be elicited on the right with reenforcement, and there is a partial return of sensation in the right foot.

We believe the symptoms to be due to some toxic insult to the peripheral nerves, probably with root involvement. That the sensory symptoms and the chief motor symptoms are both on the same side, that the sensory symptoms on the right side are of the stocking type, that the spinal fluid is normal, that the condition of the patient is improving, and chiefly the fact that there are electrical changes of a quantitative type, all urge this diagnosis, to the exclusion of another, which perhaps the single finding of nystagmus would lead us to, namely, multiple sclerosis. We feel that the case has a good prognosis with the former diagnosis. The coordination is very good. During the early stage he had slight pain in the legs, but it was very indefinite, and for the most part he has not had true neuritic pain.

CASE OF GASOLINE OR GAS POISONING. Dr. W. M. Kraus.

The patient had been working about a gas engine, exposed to the fumes from the exhaust. He started at that particular job in August, 1920, and entered Bellevue Hospital in June, 1921. At that time he was troubled with numbness, weakness and coldness in the legs, hands and forearms. This trouble had begun the previous February, when he complained of dizziness, some nausea, frontal headache and general malaise. He had had no stomach attacks before that. His legs slowly became weaker, so that by April he could not walk. He had no other symptoms.

Examination revealed that his cranial nerves were normal. He had a bilateral hypesthesia of a stocking variety almost to the knees, and in the toes a slight defect in sense of position. He was unable to stand. Six blood Wassermann tests and three spinal fluid Wassermann tests were negative. The last blood Wassermann test was followed in about three weeks by administration of arsphenamin.

At present he shows marked atrophy of all extremities. He has no more fibrillary twitching. His superficial reflexes are still present. He shows no signs of involvement of any of the other nerves.

Thompson in his book on "Occupational Diseases" states under the heading "Gasoline Poisoning":

> Lately there have been brought to my notice a number of cases of chauffeurs who were overcome by gasoline fumes, while cranking motor cars in small, unventilated garages. They experienced vertigo, fainting and nausea, and one passed into coma from which it was difficult to arouse him. The symptoms may not develop until the men go into the open air.

About three years ago three men in one garage complained of the same symptoms, and they told me at the time that this disease was not infrequent among chauffeurs. Since then I have had another case, but it has not shown the profound atrophy found in this case.

DISCUSSION

Dr. Kennedy: Perhaps Dr. Kraus did not see a paper by Gowers about fourteen years ago. Gowers had a group of gasoline poisoning cases very much resembling vasoneurosis, but without any involvement of the nervous system as such.

Dr. E. G. Zabriskie: My impression of this case is that the poison came from the exhaust, as in most other reported cases. Therefore, it is carbon monoxid poisoning, and it is incorrect to speak of it as gasoline poisoning.

EVIDENCE OF NERVOUS CONTROL OF SOME INTERNAL SECRETIONS. Dr. Walter B. Cannon.

Dr. Cannon of the Medical School of Harvard University (by invitation) presented this paper illustrated with lantern slides.

He said: My interest in the control of internal secretion began years ago in the observation that emotional excitement inhibits the movements of the stomach and intestines. This inhibition is due to sympathetic impulses. The question arose as to whether the suprarenal medulla, which is also innervated by the sympathetic, and whose output, epinephrin, mimics sympathetic action in the body, is likewise caused to secrete during emotional stress.

Evidence of Nervous Control of the Suprarenal Medulla.—The first evidence was given by de la Paz and myself in 1911. An intestinal strip rhythmically contracting in oxygenated Ringer's solution is promptly inhibited by very minute amounts of epinephrin. It is likewise inhibited by blood removed from the inferior cava in the neighborhood of the lumbosuprarenal veins, if the blood is taken during or immediately after excitement, but is not inhibited by blood taken from the same region before excitement. The conclusion was, therefore, that the suprarenal medulla discharges into the blood stream its peculiar product, epinephrin, in periods of emotional stress. Later Hoskins and I showed that asphyxia and afferent stimulation, both of which are known to cause discharge of sympathetic impulses, would likewise induce extra suprarenal secretion. The evidence thus adduced has been confirmed by various observers using various methods.

The results and the methods leading to the conclusions just mentioned have been criticized by Stewart and Rogoff. They have declared that the output of epinephrin from the suprarenal glands is not increased by asphyxia, by reflex stimulation, or by excitement; indeed, that the secretion is constant and unchangeable. Furthermore, they urge that it is impossible to judge whether the secretion is increased or decreased unless the rate of blood flow is known. I shall now defend the method that we employed, though it can be defended by use of recent evidence that the blood flow in the inferior cava is, in fact, increased under the conditions cited, so that the tendency would be not to concentrate a constantly secreted epinephrin but rather to dilute it; in other words, the conditions of blood flow are unfavorable to the result which de la Paz, Hoskins and I actually found. Instead of elaborating this line of defense, I wish rather to speak of the use of the denervated heart as an indicator of suprarenal secretion. In this preparation both vagus nerves are severed, the stellate ganglions are removed, and the heart itself, separated from the central nervous system, remains in the body performing its function, but no longer subject to any influences except those brought by the blood stream. The heart thus isolated is extraordinarily sensitive to circulating epinephrin. Though very stable in its rate, it can be made to beat faster by 30, 40 or 50 beats per minute, by inducing asphyxia or reflex stimulation or excitement. Such increases, in our experiments, did not occur or were relatively slight when the suprarenal glands were removed. The same conclusion was drawn, therefore, that was drawn from the earlier experiments, that the three conditions just named induce a secretion of the suprarenal medulla.

Again, the evidence has been criticized by Stewart and Rogoff. They repeated the experiments with the denervated heart and found that in certain instances they were able to obtain a marked increase of rate though the suprarenal glands had previously been removed. Examination of their results showed that the order of increase of the two conditions was as a rule very different. The heart rate is on the average increased approximately 30 beats per minute if the suprarenal glands are intact, whereas the increase is only 6 beats per minute after their removal. Nevertheless, there are instances of much larger increases than the average of 6 beats, and these cases must be accounted for.

In experiments in cooperation with Rapport, I have found that the increase cannot be explained by increased blood pressure, arterial or venous, by warmer blood from the abdomen, or by accessory suprarenal tissue. It disappears, however, on severance of the hepatic nerves. For the present we may leave the matter at this point and understand that after cutting these nerves reflex stimulation or stimulation of an afferent nerve will produce a pure suprarenal effect.

In explanation of the faster heart rate when the suprarenal glands are present and an afferent nerve is stimulated, Stewart and Rogoff have suggested that such stimulation by constricting the splanchnic area induces a "redistribution of blood in the body," with consequent concentration of the uniformly secreted epinephrin which thereby becomes more effective. This suggestion we have put to test. By use of a "reduced" animal (that is, one with both carotid, both brachial and both renal arteries tied, the aorta closed below the renals and the mesenteric nerves cut) vascular reflexes are reduced to a minimum. The liver is excluded and the suprarenals are the only abdominal viscera left innervated. Typical reflex accelerations of the denervated heart averaging 26 beats per minute occurred in this preparation, although the changes of blood pressure were insignificant or were only moderate variations either up or down. Under these circumstances the more rapid beat cannot be attributed to a redistribution of blood in the body, for the possibilities of redistribution were lacking.

Another test consisted in substituting a stream of epinephrin to replace suprarenal secretion after removal of the glands. In this test epinephrin at the rate of approximately 0.0007 mg. per kilo per minute were injected by vein continuously to replace the continuous unvarying suprarenal secretion assumed by Stewart and Rogoff to be normal on the basis of a test made inside the body. Typical vascular reflexes were now induced by afferent stimulation, but either no acceleration of the pulse occurred or an insignificant increase of two beats per minute. Again the evidence is clear that the faster rate is not due to redistribution of blood in the body.

Stewart and Rogoff have urged that their method is superior to all others in being quantitative. We have found that the denervated heart in the "reduced" animal can be employed as a means of assaying epinephrin. Repeated equal doses injected intravenously at a uniform rate repeatedly produced in a given animal the same or nearly the same maximum increases of heart beat, and if the injections differed in rate the increases likewise differed in degree. When the circulation is thus simplified the acceleration is wholly or chiefly dependent on the rate of injection, and is little, if at all, influenced by variations in blood pressure. By matching epinephrin injections with reflex epinephrin secretion we have shown that when the heart rate is accelerated between 30 and 42 beats per minute, the output from the suprarenal glands lies between 0.0032 and 0.0037 mg. of epinephrin per kilo per minute, i. e., more than ten times the amount regarded by Stewart and Rogoff as the unvarying normal secretion, when they removed blood and assayed it for epinephrin outside the body. Stewart and Rogoff have not explained the discrepancy between their figures for the internal and the external test for suprarenal secretion.

From the foregoing evidence we conclude that the results of our earlier work have been verified and that the inferences drawn from it that suprarenal secretion is evoked by asphyxia, by reflex stimulation and by excitement, are justified.

Evidence for Nervous Control of the Liver.—It will be recalled that the denervated heart may be made to beat faster on afferent stimulation though the suprarenal glands have been removed, and that this effect is abolished by severance of the nerves to the liver. The inference was drawn, therefore, that some agent arising in the liver is the occasion for the faster rate. Uridil, Griffith and I have shown that stimulation of the hepatic nerves themselves will cause an increased rate of the denervated heart, an effect appearing slighty later than the similar epinephrin effect and lasting for a longer time. Furthermore, stimulation of the hepatic nerves will cause a rise of blood pressure. This is not due to a retarded blood flow through the liver, for it does not occur on closure of the hepatic artery and vein. Furthermore, it does occur on hepatic stimulation though all the abdominal viscera have been removed except the liver, and unlike the stimulation of splanchnic blood vessels alone it long outlasts the period of stimulation.

The increments of heart rate vary widely. They are slight if the animal has been fasting or is in poor condition. They are much greater if the animal is digesting meat. These conditions can account for the discrepancies between Stewart and Rogoff's results and those reported by myself and my collaborators as a consequence of splanchnic or reflex stimulation after removal of the suprarenal glands. The inference is drawn, therefore, that a substance is given off by the liver into the blood stream which, carried to the denervated heart, raises its rate.

None of the known or supposed products of hepatic activity—glucose, urea or catalase—when injected into the blood stream have the effects produced by exciting the hepatic nerves. Furthermore, the efficacy of hepatic stimulation in causing a faster heart rate when meat is being digested is not seen in an animal which is digesting carbohydrate or fat or which has been fed for several days on either of these foodstuffs. On the other hand, stimulation is more effective after amino acids have been injected into the intestines.

For the present we are inclined to draw the conclusion that probably the effects noted are not due to a true internal secretion produced by the liver, but to a discharge from its cells of amino acids or amins which are sympathomimetic in character.

Evidence of Nervous Control of the Thyroid.—Histologists have described nonmedullated nerve fibers reaching to the cells of the thyroid gland. Anatomists find that fibers going to thyroid gland arise in cervical sympathetic ganglions. It had been shown that severance of its cervical sympathetic nerves would cause atrophy of the thyroid and that stimulation of these nerves would cause a diminished iodin content of the gland. In 1916, in cooperation with Cattell, I pointed out that if the thyroid gland and neighboring indifferent tissue are connected through a galvanometer, stimulation of the cervical sympathetic cord evokes an action current after a latent period varying between 5 and 7 seconds, an effect which persists after the superior and the recurrent laryngeal nerves are severed. This does not occur if the main trunk of the vagus is stimulated, or if pilocarpin, which is an excitor of vagus endings, is injected. The effect cannot be attributed to the shutting off of the blood supply by vasoconstriction, for total anemia produced by clamping the carotids for a period equal to that of sympathetic stimulation produces no noteworthy electrical changes in the gland. From this evidence the conclusion was drawn that the nerve fibers distributed to the thyroid cells belong to the sympathetic and not to the cranial division of the autonomic system, and since their effects are not indirect through alterations of blood supply, they are indeed true secretory nerves.

The electrical evidence given in the foregoing was confirmed by the experiments of R. L. Levy in the Harvard Physiological Laboratory in 1916. He made use of the fact that if thyroid substance is introduced into the blood stream standard doses of epinephrin injected intravenously will cause a greater rise of blood pressure than they caused previous to the thyroid dosage. He found that after stimulation of the cervical sympathetic there was indeed a greater effectiveness of the suprarenal injections amounting in some instances to as much as 200 to 300 per cent.

Since epinephrin will mimic the action of sympathetic impulses it was to be expected that epinephrin itself would stimulate the thyroid. This Levy found to be the case. Stimulating doses of epinephrin caused a greater effectiveness of the repeated standard doses (which were very small). If the thyroid glands had previously been removed, however, both cervical sympathetic stimulation and epinephrin injections failed to produce an increase of the response to the standard doses.

Levy proved that stimulation of the cervical sympathetic caused the thyroid to secrete promptly. Removal of the thyroid gland immediately after stimulation was followed by an increase of the efficacy of the standard doses similar to that observed when the glands were left in the animal throughout the course of the experiment.

The method used by Levy involved destruction of the upper part of the central nervous system. This procedure did not permit tests to be made on the effects of such stimulation as would in the conscious animal be accompanied by sensations of pain, nor on the influence of asphyxia. It was desirable to have another method, therefore, which would permit such tests as these to be made.

In 1920, in cooperation with P. W. Smith, I made use of the denervated heart as an indicator of thyroid activity. If the temperature of the animal is kept uniform and the ether anesthesia is likewise maintained at a fairly uniform level, the heart rate does not vary more than a few beats a minute through several hours of observation.

With the denervated heart we found that gentle massage of the thyroid gland for two or three minutes would cause an increased rate amounting in some instances to 33 per cent. over the basal rate. The development of the maximal increase was usually slow, requiring from 30 to 60 minutes and passing off in a similarly slow manner. Massage of another gland, for example, the submaxillary, did not cause this effect, and massage of the thyroid would cause it even though the suprarenal glands had previously been removed.

We found that stimulation of the cervical sympathetic trunk would induce a similar increase of the rate of the denervated heart, but that this did not occur if the thyroid gland had previously been removed on the stimulated side. Reflex stimulation of the sciatic or brachial nerve, such as would cause sympathetic impulses to be discharged, had the same effect as direct stimulation of the cervical trunk, but with the addition that there was a primary acceleration of the heart due to suprarenal discharge. This was followed by the same slowly developing thyroid effect. Asphyxia, likewise, had the same influence. On the other hand, if the thyroid glands had been previously removed sensory stimulation and asphyxia induced only the increased rate due to suprarenal discharge.

The foregoing evidence—electrical, cardiac and vascular—is harmonious in testifying to a control of the thyroid through the sympathetic strands in the neck. By connecting the phrenic nerve with the cervical sympathetic and waiting until the phrenic fibers have grown out to the distribution of the sympathetic fibers we have been able in a certain percentage of cases to produce many of the phenomena of hyperthyroidism. When this operation is done, there would be discharged to the distribution of the cervical sympathetic nervous impulses with every respiration. Thus, nerve filaments affecting the thyroid would be continuously excited by normal impulses from the central nervous system. In animals which showed indications of hyperthyroidism there was an increased heart rate running up from the normal, which in the cat is approximately 150 beats per minute, to 225 and even 250 beats per minute. The character of the animal changed from that of a quiet affectionate domestic pet to that of a furtive creature running away from any one entering the room. There were loose movements of the bowels without evidence of fermentative changes. The skin of the neck and head became thickened in spots, and there were patches of alopecia. The most characteristic alteration, however, was a great increase in metabolism. In one of the animals the increase of the metabolic rate was 125 per cent. Removal of the thyroid gland on the operated side restored the animal in which the metabolism had been greatly increased to a rate which was within the normal range of variation.

The Nature of the Nervous Control.—Thus far evidence has been adduced that suprarenal secretion, an output of a stimulating material from the liver, and likewise thyroid secretion, are all subject to sympathetic impulses, that is, to the activity of that part of the nervous system which is roused in great excitement. It is important to know where the reflex center for this activity is and whether the control of the organs mentioned is double as in the case of the submaxillary gland (chorda and sympathetic), or single like the biceps, for example, with inhibitory or excitatory impulses affecting the central station.

Answers to these questions have been found for the suprarenal medulla in experiments performed by Rapport and myself. We have observed that reflex acceleration of the denervated heart can be evoked after removal of the cerebrum and the corpora quadrigemina. A transection a few millimeters back of these bodies destroys the reflex.

Removal of the suprarenal glands (without a fall of blood pressure) lessens the rate of the denervated heart. Under experimental conditions, therefore, a continuous suprarenal discharge is going on. Using this as a basis we have regarded slowing of the rate of the denervated heart in consequence of stimulation as being due to diminished suprarenal secretion. Such slowing occurs when the central end of the vagus or of the depressor nerve is stimulated in the cat. It may be as much as 24 beats per minute and appears even after the corpora quadrigemina and all parts anterior to them have been removed. It fails to appear when the brain stem is severed a few millimeters back of the corpora and likewise after the suprarenal glands have been exercised. We conclude, therefore, that the reflex center for suprarenal secretion is located near the upper or front edge of the floor of the fourth ventricle and that it is subject to both excitatory and inhibitory nervous influences.

The evidence just adduced places the control of suprarenal secretion in a part of the primitive nervous system. It is interesting to note that this region is in close approximation to that which appears to be concerned in emotional expression. Sherrington and Woodworth were able to call forth pseudoaffective reflexes in the cat after ablation of the hemispheres and the thalamencephalon, and Goltz likewise noted many evidences of emotional expression (snarling, snapping, etc.) in a dog from which the cerebral cortex had been removed. Head and Holmes, because of clinical studies, were led to the conclusion that the thalamus in man is concerned with the affective side of sensation. It appears, therefore, that the emotional reactions of animals are not managed in the neopallium but are ingrained in an archaic portion of the nervous system. Possibly it is for this reason that the expression of emotions is so similar in widely different animals, such as the cat or the dog, and man. The fact, however, that such is the case permits us to use the lower animals in an analysis of conditions accompanying emotion outbreaks and justifies the expectation of finding similar conditions and a similar analysis in human beings. The great interest of study along lines here hinted at lies in the association of primitive and archaic reactions occurring in an ancient portion of the nervous organization of the body and operating on an elementary form of the nervous system (the sympathetic) with the functions of organs which are fundamentally important for the organism—the ductless glands.

DISCUSSION

Prof. Frank H. Pike of Columbia (by invitation): It is a peculiar pleasure to me to discuss this paper, for two reasons: For many years Dr. Cannon and I have been friends; that is the first reason. In the second place, it is not very often that physiologists get a chance to talk to neurologists.

It has long been recognized that the production of an electrical current is an invariable accompaniment of the processes of living matter. Walter has gone so far as to characterize the production of electrical current as a sign of life.

Whenever a gland secretes, whenever a muscle contracts, there is an electrical current produced; and today we make wide clinical application of the fact that the heart produces an electrical current; that this current can be led off and its variations measured with precision. We judge the minute changes in the heart merely by its electric cardiogram.

There can be no question, I think, that a change in the electric condition of a gland or muscle indicates some change in its degree of metabolism. One may object that the changes are small; that there may be other tissues in that gland aside from gland cells, and it is not always easy to separate the gland cells from the other tissues. The objection probably has some weight; but I think, taking into consideration the facts that go along with it, that there is some nervous control of such an organ as the thyroid.

With regard to the suprarenals, I am frankly puzzled. I did not come here with the intention of saying anything about our own results. It seems strange that Dr. Cannon, beginning with a study of the intestine, should find that the root leads back to the nervous system. It seems perhaps strange that for myself, beginning with the study of the central nervous system, I should somehow or another get into this suprarenal mixup. Yet, such is the case.

I do not care to say much about interpretations now; I am frankly puzzled. But, if you will pardon me, I will depart from my original intention and briefly describe some things which bear on this question.

Some years ago Professor Stewart and I (this makes matters worse) tied off the blood vessels to the head of a cat. We obtained a very vigorous response. We obtained a great variety of blood pressure, we obtained changes of heart rate, we obtained changes of respiration, and the whole picture was rather characteristic. While about two or three years ago, possibly four, Mrs. Lincoln set about analyzing the vasomotor pathway more in detail—we wanted to find out where these vasomotor fibers came out; we wanted to find out, first, whether it was the vasomotors that were concerned. We believe now that those vasomotor fibers are concerned. But rather a peculiar thing was noticed. This tying off of the blood vessels to the brain, of course results in total failure to function. After a time the animal lies as if dead. If you keep up artificial respiration, the heart and the entire other part of the system is open, so that the head is the only part deprived of oxygenated blood. It does not do the cat much harm to kill it; in fact, we kill the cat several times. We find that this procedure of tying off the blood vessels for three or four or five minutes, whatever is necessary (allowing this whole change of blood pressure, heart rate, whatever else it is that happens, to go on, until there is a failure of the function of the medulla), restoring of circulation, getting the cat back again, can be repeated as often as eighteen times. The cat seems to be very resistant. Yet, to all appearances, this is a pretty severe procedure.

It was noticed, however, that as the cat was subjected to these successive insults, that the blood pressure curve began to show some differences. We had at first simply the curve rising, a little dip in it, and another rise, and another fall. It was plainly enough one curve. But after these blood vessels had been tied off, say, ten or twelve times, or eight or nine times, depending on the cat, we began to find a notch in the top of that curve; there was a partial drop in that blood pressure, then it came back again, and we obtained a final fall. Toward the end of this series we found that the blood pressure rose pretty sharply and fell perhaps way down to its normal level and again rose. We obtained this curve breaking up in two; it was pretty symmetrically divided in point of time, but a repetition of this process for three hours did not suffice to kill the cat.

The chance observation that the blood did not clot very readily in such a cat made us suspect that the suprarenals might be involved here, so we tied off the suprarenals. There is no particular change in blood pressure when this is done, but the picture subsequently is rather different. In some cats, immediately after typing off the suprarenals, simply tying off the blood vessels once is sufficient to curtail this response by about 50 per cent. We get simply the sharp rise and the blood pressure falls, say, within half the time occupied by the control. Sometimes four or five conclusions are necessary, but invariably the animal dies; we find that it is impossible to bring it back, after this procedure has been employed a few times. There is something in that epinephrin which is extremely necessary for this relationship between smooth muscle and sympathetic nerve. I have an idea that without the suprarenals, neither people nor animals would be able to stand nearly as much as they do in this world. It might be better for some of us if we could.

It seemed to us rather important that we could shorten this survival time of an animal after the removal of its suprarenals, say, to a period of fifteen minutes and not have to wait twelve or twenty-four hours for it to die. The longest time any of them have survived is a day and a half. There are so many of them, I am not perfectly sure of the figures, but the time was of that order of magnitude.

That is about as far as we have progressed, and, thinking the matter over, I can see that there is enough to keep me busy probably for the next generation, working out some of these matters in detail, but I suspect that somewhere in our work there are conditions which we have not yet taken into account, which are causing these discrepancies. Where they are. I do not know, but I do not feel like talking about them now. The safest way by far, I think, is to go back to the laboratory and interview the cat about it. The response, I think, would be far more trustworthy than anything I might say.

Again I must express my pleasure at the presentation Dr. Cannon has given us, and if we disagree in matters which seem to you trivial, mere squabbles among ourselves, I hope you will forgive us. The thing promises some interesting discussion somewhere, until we run down these conditions which lie at the bottom of our discrepancies.

Dr. Walter Timme: The general proposition, I venture to think, is that the physiologists, while they are rather accurate in the work they do, nevertheless do not agree among themselves as to observations or conclusions even from the same observation. I do not think that we are very much worse off than they. We travel, perhaps, a little faster; I do not know that we get much further. And so the subject is left more or less as Dr. Pike left it, and that is as far as we get.

To me it seems that Dr. Cannon's work is not destructive but constructive. Stewart and Rogoff seem to be quite destructive. So that for the present, and I hope for the future, I and all of us will be able to bear with Dr. Cannon in his further work.

I should like to ask Dr. Cannon if stimulation of the sympathetic nervous system or a portion of it, or of a peripheral nerve, will produce an increase in the suprarenalin content of the blood. Then, if it is granted that epinephrin in the blood stimulated the sympathetic nervous system (and I think that has been proved), we are immediately confronted by a condition of unstable equilibrium in which epinephrin stimulates the sympathetic, the sympathetic stimulates the epinephrin production, and so we go on with a constantly increasing production of epinephrin until finally, if the production went on ad infinitum, exhaustion would ensue. There must therefore be an inhibitory center which automatically must bring that process to cessation, or else we would all succumb; as a matter of fact, we do in certain instances succumb to the stimulation of the suprarenal medulla by means of thyroid activity. The administration of thyroid in a susceptible person will produce syncope, due, in all probability, to exhaustion of the suprarenals through that administration.

Dr. Cannon has explained to us that the center for these reactions is probably in the thalamic region. That perhaps is the old brain center of affective motor control. There must be a higher center in the brain which, through its stimulation and its emotions, controls this lower center, and, although Monakoff has not definitely pointed out such pathways, I have wondered whether since his time physiologists such as Cannon have had any experience in their laboratory with the physiology of those pathways.

Dr. Pike made the statement, which had been made previously a number of times, that the activities of all organs, and especially of the internal secretory organs, could be enhanced or produced not only by one method but by many. That leads me to this statement: A great deal of the outpouring of epinephrin and of thyroid secretion is perhaps not due to nerve current at all but to direct massage, just as he explained, experimentally, it could be produced. Deep breathing, ordinary respiration, with the movement of the diaphragm downward, compressing the abdominal organs, in itself has the mechanical and physical effect of producing and of releasing into the circulation a certain amount of epinephrin so that massage (physical causes, as well as electrical ones) will suffice to produce the secretion.

Then there is a third one, a direct chemical effect, as has been shown a number of times, I believe, especially in the frog's heart removed from the body. A frog's heart removed from the body will beat rhythmically for a certain number of hours if properly maintained in the proper fluids; but if a little thyroid solution be added to that, the number of hours will be enormously increased. The direct chemical effect, therefore, besides the physical, besides the electrical, is an element of the production of the internal secretory flow into the circulation.

Dr. Israel Strauss: Dr. Timme has stated that in the cases of certain persons if you administer large doses of thyroid, you may get syncope, and that that is due to extra excretion of epinephrin, due to exhaustion of epinephrin. I want to ask Dr. Cannon if there is any physiologic proof of that. Dr. Cannon has shown on his charts that when epinephrin is introduced, the activity, through thyroid, is increased. I believe that is what the chart shows. But I believe he has not shown us whether increased administration of the thyroid has any effect on the suprarenal.

It is just such physiological facts that are necessary to us clinicians.

I am very anxious to know whether Dr. Cannon has proved, or if he can quote work which will show definitely, that the thyroid secretion given to an animal can influence the suprarenal glands as he has shown that the suprarenal gland can affect the thyroid gland.

Dr. Stewart Paton of Princeton (by invitation): I think probably Dr. Cannon does not need to be reminded that we have a very excellent opportunity of studying these archaic reactions in the embryo of all the higher vertebrates. I feel that my opportunity has come, because I have been repeating for a number of years that somebody with Dr. Cannon's capacity for conducting experiments should try to correlate the functions of the embryo in relation to the development of the nervous system.

As I have said before, first the thyroid comes into the circuit and then the suprarenal; and I was very much interested to hear Dr. Cannon speak about the abundant supply of the suprarenal. That is my feeling, and still I would like to keep the question open.

I was also glad to hear him refer to the center for the nerves that supply these organs, the corpora quadrigemina in that region. From the long fasciculus which develops in the embryo you can trace the nerve supply down to the thyroid and you can trace that fasciculus, getting down very much later to the suprarenals, at least comparatively speaking; so that the thyroid comes into the circuit quite a good deal ahead of the suprarenals, and it seems to me that if we observe the embryo very carefully at this time, a good deal of information can be obtained in regard to the functions of the thyroid and the suprarenals in relation to the nervous system.

I was glad to hear Dr. Pike say a word about the nervous system, because I know a physiologist whose attitude toward the nervous system is very similar to that of an English clergyman I met, who asked me what I was working at. I said, "The brains of a fish." He said, "Do fish have brains?" I said, "Yes." He said, "By Jove, that's quite an idea!"

Dr. Cannon: Dr. Pike's description of the discharge from the suprarenal gland, the evidence at least in the experiments that he and Stewart did together, that there is a storage in the suprarenal gland when the blood supply is cut off from the medulla, seems to me to fit in with all the evidence I have presented in regard to the suprarenal, in regard to the nerve supply, causing a rise in blood pressure that is well known and developing cerebral compression, for example, and would therefore cause a discharge of impulses similar to that which was illustrated this evening, and a discharge which would result in an increased output of epinephrin from the medulla and a consequent rise of blood pressure—that rise with a dip that was pointed out two years ago in an observation that was made in the Harvard Laboratory, and was also noted by Elliott, and later worked out in more detail by Vincent. That disappeared, you will recall, in Dr. Pike's experiments, after the suprarenal glands were tied off. In those experiments, after the suprarenal glands were tied off, the animals, he said, died sooner than was the case when the glands were not tied off. Now he drew the conclusion that that indicated the importance of the suprarenal medulla. I do not believe that he is really justified in drawing that conclusion because when he ties off the suprarenal glands, he ties off two glands of the suprarenal medulla and cortex.

Then Dr. Timme raised a very large question as to whether the physiologists had any advantages over the clinicians as workers in the realm of science. I think I am not perhaps misstating his remarks; at least, he suggested that inasmuch as there are differences of opinion among clinicians and also differences among physiologists, that they are both on the same plane in that they have differences; but it seems to me that the physiologists at least have this advantage over the clinicians: they work on material and concern themselves with problems which are capable of demonstration and capable of being inquired into under conditions which are more or less controllable.

For instance, take the question that Dr. Strauss referred to in Dr. Timme's remarks, the question whether syncope is due to exhaustion of the suprarenal because of feeding the thyroid. Now, it seems to me that there are a number of wholly unwarranted assumptions in that statement. In the first place, it is possible to remove both suprarenal glands from animals, and they will live for hours. We have had them live for three or four days, with no drop of blood pressure whatever. It is a very common assumption, that the suprarenal medulla is necessary for the maintenance of blood pressure. Now, that is not at all a demonstrated fact. In all probability the fall of blood pressure that occurs finally after removal of both suprarenal glands is a consequence, possibly, of the removal of the suprarenal cortex rather than the medulla; a general weakening of the whole animal. At least, it is possible to remove practically all of the medulla from the suprarenal glands, leaving the cortex, and the animals will survive for a long period; and then on removing whatever is left, they promptly succumb. I do not know of any evidence that the feeding of the thyroid produces any exhaustion whatever of the suprarenal medulla; in other words, the feeding results in an increase in the size of the suprarenal glands, and, as I showed you this evening, that involves both the cortex and the medulla. So that we are at least capable of trying out these matters and getting at the evidence under conditions which are, I think, more largely under control than is the case in practice on the human being.

So far as the difference between Dr. Stewart and Dr. Rogoff and myself is concerned, that is a matter of getting out the evidence. After this evidence was obtained, I was kindly invited to go to Cleveland and speak to the Cleveland Academy of Medicine, which I did in May, and presented this material, and Dr. Stewart listened to it and did not offer any constructive criticism, and I do not see that he has very much to do except to find out now what is wrong about his own experiment.

As I told you earlier, most of this material is not yet published. The work on the suprarenal gland will come out in the December number of the *American Journal of Physiology,* and I hope that when that evidence is before investigators it will be convincing. I am ready to demonstrate any point that I have made here this evening, at any time, to any group of physiologists or other interested people, so that if there is any question in any of your minds as to whether this evidence is on the same basis with clinical evidence, you can take at least that method of discriminating between two types of evidence.

There is another point that Dr. Timme brought up, and that is why we did not get into a regular cyclone of sympathetic activity because the sympathetic stimulates the suprarenal, the suprarenal stimulates the sympathetic, and so the circle goes, until we wind up in a catastrophe. Well, that might occur if the suprarenal gland were subject to suprarenal secretion, but there is no evidence that it is, and there is quite a bit of evidence that it is not. It has a peculiar nerve supply; it is not enervated by the sympathetic as the blood glands are enervated by the sympathetic. So that the possibility of a vicious circle being started is not present, and, as the record showed this evening, after an afferent nerve is stimulated or the splanchnic nerve is stimulated, the heart rate goes up promptly, beginning about eleven or twelve seconds, and in perhaps a minute and a half after the stimulation has ceased, the heart rate is down where it was before. In other words, this vicious circle does not occur, and it does not occur because the secreted epinephrin does not in turn stimulate the secreted cells to produce more epinephrin.

With reference to a higher center for emotion than those which are found in the thalamic region, I should like to have more evidence. It seems to me that the observations of Head and Holmes were extremely interesting observations, and if you will recall that the study of their cases indicated that these persons had an exaggerated affective accompaniment of all sensation, just in so far as the evidence indicated that this thalamic center was disconnected from control by the cortex, and they suggested that here was a mechanism in the thalamic region already for expression of affective states of emotional experience and that it was inhibited by the cortex, and that when the cortical inhibition was removed, this lower center then had full freedom of expression. The experiments of Sheridan and Woodworth and also the observation of Goltz indicated likewise that this lower mechanism was quite adequate at least for the expressions of intense emotion such as rage, or possibly fear; certainly the aspect of attack is shown by these animals quite apart from any play of the cortex, because the cortex has been wholly removed.

Book Reviews

PSYCHOLOGIE DER WELTANSCHAUUNGEN (PSYCHOLOGY OF WORLD VIEWS). By Karl Jaspers. Pp. 428. Berlin: Julius Springer, 1919.

The work claims to be an attempt, "only to understand what final positions the soul assumes and what forces move it." The author realizes the weighty objections that may be made to his venture in this field, but justifies his efforts by the conviction that his presentation in orderly form of whatever has already been recognized as psychology of "Weltanschauungen" would in itself offer something of value. Hegel, in the "Phenomenology of the Spirit," is the author's only great predecessor, but Jaspers differs in not erecting a uniform, systematic structure but in presenting a schematic arrangement, not following one method like Hegel, but now this, now that one. Kant, Nietzsche and Max Weber offer much in support of the view of the author, as does also the Dane, Kierkegaard, whom he regards with great favor.

Jaspers would divide the whole psychology of "Weltanschauungen" into three parts: (1) general—basic principles, limits, forces, etc.; (2) the spheres of personality, of activities like science, metaphysics, art, religion, etc., and the social sphere, applying to a greater or less extent the categories of Part 1; (3) the most concrete and actual part, consisting, because of the mass of material, in studies of single personalities, peoples, ages, conditions, or professions, etc.

Jasper's present work is limited to the first of these parts: general principles presented in an attempt at order—at completeness of principles, if not of material. It consists of three long chapters with subdivisions. Chapter 1 is on the various standpoints (Einstellungen, "attitudes," "adjustments," "focusings"): objective (active, contemplative, mystic—all with subclasses); enthusiastic (covering self-sacrifice, love, creative impulse, etc., with a few words on ecstatic conditions and intoxication). Chapter 2 treats varieties of the "Weltbild," which is, as it were, the shell in which we live and which limits our world for us—"we do not see what lies beyond for we do not even suspect its existence." Here again there is a threefold division: the material-spatial, the psycho-cultural and the metaphysical worlds. Within the last group Jaspers presents material from Goethe, illustrating that author's conception of the "demonic" element in his world; in another section there is a discussion of the types of philosophical thinking (illustrated by the Greeks). The last chapter is on "types of mind," under which term "principles, ideas, forces, are understood"; the effort is made to bring these directly into the field of vision and, by reversing the process of the first two chapters, to trace their manifestations in "Einstellungen" and "Weltbilder." Tables of values are suggested and discussed (the highest good, ultimate aims, etc.); the antinomic structure of existence, optimism and pessimism, skepticism, etc., nihilism in psychoses (pp. 265 to 269) Jasper illustrates by a case which he reported in 1913 in the *Zeitschrift für die gesamte Neurologie und Psychiatrie*. In appropriate places in all parts of the work the author inserts in fine print summaries of the pertinent thought of various philosophers; in the last chapter ten such pages are devoted to Kierkegaard. There is a twenty-page appendix on Kant's doctrine of the idea.

A full table of contents only partly makes up for the lack of an index.

Jaspers does not pretend to give a direct answer to the concrete questions of actual life; his book makes an appeal to the "free intellectuality and activity of life by offering means of orientation, but it does not attempt to create and teach life" (preface). The volume represents a most serious effort to handle a large subject. It is possible that in a later edition the author will find occasion to rewrite, revise and clarify many sections of the work. It is not a book which a beginner in philosophical study would pick up, or profit much from if he did. Only those with much experience in philosophical reading in German are advised to peruse the work—it is not merely the occasional pleasant compound like "das Sichdurchsichtigwerdenwollen" (p. 94) that he will have to swallow.

THE NERVOUS CHILD. By Hector Charles Cameron, Physician to Guy's Hospital. Pp. 202. London: Oxford University Press; American Branch, 35 West Thirty-Second Street, New York, 1919.

This little book so handy in size and so well printed, as is apt to be the case with English books, on lightweight paper, contains a modicum of common sense. The author defines his work as an introduction to the teachings of psychology, physiology, heredity and hygiene as applied to medical work in nurseries and in schools. But it goes beyond any introductory standpoint; it is in places, especially in therapeutic recommendations, baldly concrete and specific.

Nervousness, to Dr. Cameron, seems to mean anything and everything. As a matter of fact, "The Nervous Child" is for him a thread on which he strings many discrete ideas—imitativeness, the child's love of power, the reasoning of childhood, sex teaching, etc. As a matter of fact, actual nervous symptoms and tendencies, such as habit spasm, hysterical phenomena, sleeplessness, receive the most scant treatment, even by way of an introductory statement.

There is a deal that might be said concerning Dr. Cameron's therapy: Hypnotism is one of his main standbys. We read that in inveterate cases of enuresis hypnotic suggestion is always, "I think," successful. Concerning chorea: "Hypnotism is often almost instantly successful, but, apart from hypnosis, curative suggestions proceeding from the attendants form the principal means at our disposal." And there are many other citations which might be made that seem curious to the reviewer.

But there is a background of extremely good sense which the author has developed from his acute observations. It is evidenced by his insistence on the value of justice and truthfulness to the child, by his remarks on the value of close understanding of children, and the winning of their confidence, and the insight that is necessary for the proper understanding of childhood life. That it represents a fair introduction to the best in either psychology or medicine we seriously doubt. It leaves the reviewer still looking forward to the publication of a really good book on the functional nervous disorders of childhood.

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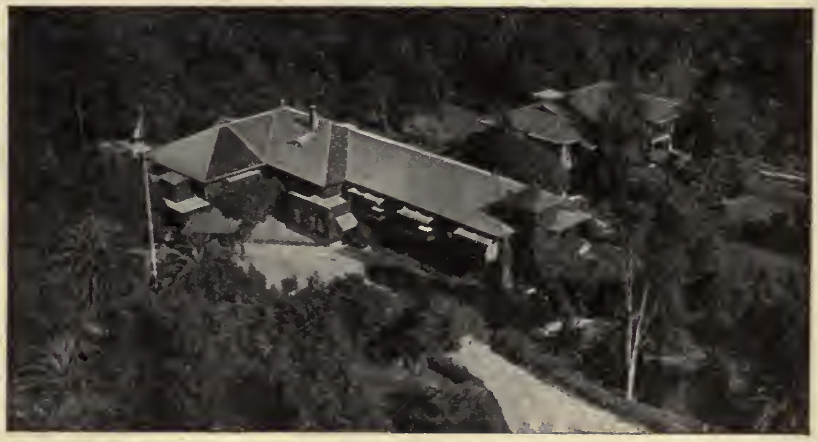

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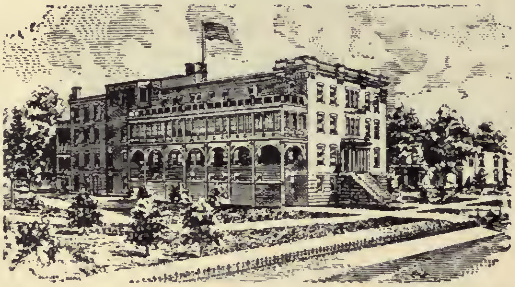

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The buildings are lighted by electricity, heated by hot water, and are well supplied by baths. The water supply for the entire institution is derived from an artesian well on the grounds, of approved therapeutic value.

The scope of the work of the sanatorium is limited to the diagnosis and the treatment of nervous and mental disorders, alcoholic and drug habituation. Every helpful facility is provided for these purposes, and the institution is well equipped to care for such patients. It affords an ideal place for rest and up-building under medical supervision. Five physicians reside at the sanatorium and devote their entire attention to the patients. A chartered training school for nurses is an important part of the institution in providing especially equipped nurses—both men and women—for the care of the patients.

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